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THE MANAGEMENT OF OBSTRUCTIVE JAUNDICE AS A FACTOR AFFECTING SURGICAL RISK*

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THE recognition of the pathological physiology underlying many conditions of which the immediate surgical aspect is only a result, has introduced a necessary pre-operative management in many conditions. The appreciation of the altered physiology and the time spent in its improvement before operation have accomplished a marked reduction of the surgical risk.

The pre-operative preparation of the diabetic and his fortification against complications, and the pre-operative attack on the dehydration and toxicity of the gastric obstruction are evidences of an appreciation of the pathological physiology of a surgical condition, the operative risk of which can be markedly improved by pre-operative medical management. The aim of this management is to prevent rather than later to meet the immediate post-operative complications.

Equally responsive but less often subjected to this principle stands obstructive jaundice. In approximately 50 per cent of all cases of jaundice absorption of bile is due to obstruction of the common duct by stones; in 20 per cent of all cases it is due to absorption of bile in the liver, or infective or catarrhal jaundice, with obstruction of the duct. From 5 to 8 per cent of the cases are due to serious infection of the gall-bladder, possibly gangrene, with or without stones. Jaundice as a result of malignancy represents 15 per cent of the cases seen; one-half of these are from cancer of the liver, th other half from cancer of the pancreas or the gallbladder and ducts[1]. Thus the subject under discussion will apply to approximately 80 per cent of all cases of jaundice.

The facts relating to the mechanics of jaundice may be briefly stated. Bile is formed in the liver and should pass freely by way of the hepatic and common ducts into the intestine. The common and hepatic ducts are joined at their point of union by the cystic duct from the gall-bladder, an added reservoir probably for the maintenance of constant pressure in the liver ducts and for the concentration and possible alteration of bile. Any causes which interfere with the passage of bile into the intestine may cause jaundice, the definitely obstructive type being caused by concretions or any obstructive material from within, and by direct closure, from external pressure by enlarged glands or new growths from without. Following the obstruction the absorption of bile depends on the presence of lymphatics in the liver, gallbladder, and ducts, and on the pressure or tension. The usual method of producing experimental obstructive jaundice consists in ligation of the common duct of the animal, but when the thoracic duct is ligated as a preliminary step, lymphatic absorption and delivery into the blood are blocked and jaundiced does not occur.

The surgical risk of obstructive jaundice is generally recognized. There are, aside from the so-called accidents of surgery, three possible fatal complications—(1) hepatic insufficiency, (2) uremia, and (3) hemorrhage. The pathological physiology serving as the background for the development of these complications consists of: (1) an insufficient liver function, (2) a toxic or "biliary" nephritis, and (3) a lack of available calcium. The improvement of these factors is the aim of the pre-operative management.

Regarding the pathological physiology of the liver, we are dealing with an organ of many functions. Because of the intimate circulatory relationship, it is affected by any blood changes occurring in the gastro-intestinal canal and spleen. This passage through the liver helps to protect the body from various toxic substances absorbed from the alimentary canal. The liver also plays a part in carbohydrate metabolism, is the most important of the organs which remove an excess of sugar from the blood, and is responsible for many changes in the intermediary nitrogenous metabolism. It contributes fibinogen to the blood, and secretes bile, which enters into intestinal digestion and the absorption of fats. Of chief interest to us now are those changes following disturbances in the secretion of bile. The bile contains three important and characteristic substances: the bile pigments, the bile salts and fatty materials, such as cholesterin.

The bile pigments are products of the breaking down of hemoglobin, the conversion taking place chiefly in the liver, although in certain

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disturbances an extrahepatic conversion of hemoglobin seems to have been demonstrated. The larger portion of the bile pigments which pass into the intestine is reduced to urobilinogen and urobilin, part of which is reabsorbed and returned to the liver, part is excreted.

The bile salts are salts of glycocholic and taurocholic acids, resulting from a combination of cholic acids with taurin and glycocoll, which arise as derivatives of protein metabolism. Of the origin of the common element—cholic acid—practically nothing is known. There is a continual circulation of bile salts in the portal circulation, between the liver and intestines, reabsorption occurring from the intestines and return to the liver taking place, after which they are again excreted in the bile. Cholesterin, another constituent of bile, is widely distributed throughout the body. Its pathological importance is due to its frequent presence in gall stones. Hence, most bile constituents are normal factors in the portal circulation, abnormal in the systemic.

The presence of these bile constituents in the systemic circulation, giving rise to a yellow or greenish yellow staining of the body, constitutes jaundice. On examination this color change is apparent in the conjunctivae, and particularly evident in the covered areas of the skin, which normally show but little color from skin pigments. During jaundice the bile pigments are usually excreted in the urine, so that the latter assumes a deep red color. In certain types of jaundice, however, such as the chronic hemolytic type, bile pigments may not be found in the urine. In the obstructive type they are constantly present. A retention of other bile constituents, such as bile salts and cholesterin, may or may not be demonstrated in jaundice.

The term "Cholemia," strictly interpreted, means the presence of bile in the blood. Of late, this term has suffered a clinical perversion to indicate any unfavorable syndrome developing in the presence of jaundice. Prominent in these syndromes are such symptoms as bradycardia, twitchings, restlessness, later somnolence and possibly coma. Whether or not these symptoms are due to the action of retained bile constituents is an open question and will be discussed later. The work of King and Stewart[2], tends to show that serious toxic effect can be produced experimentally by the injection of bile and bile salts. Yet it may be improbable that the concentration of these in the blood of jaundiced patients is ever sufficiently high to give rise to marked nervous symptoms. Complete obstruction of the common duct may persist in man for months without the intervention of serious symptoms. Animals which die as a result of a mechanical obstruction of the common duct show extensive necropsy changes of the liver parenchyma. The consequent liver changes rather than the jaundice itself may more logically be the cause of the fatal result. This conception also coincides more completely with the clinical observation that the duration of a mechanical obstruction is a criterion of the functional condition of the patient.

Pathological findings on examination of the livers of both humans and animals dying after a prolonged obstructive jaundice are consistent and definite. The liver is small and yellow, with the central portion of the lobules packed with bile thrombi. The hepatic cells are atrophied in the central zone, assuming a more normal appearance as they extend outwards to the periphery. The peripheral cells often show hydropic changes and moderate deposit of fat granules.

Because of the marked regenerative and compensatory ability of the liver, it is difficult to obtain exact data as to the progress or degree of liver failure in these cases by the use of any clinical tests of liver function. As a measurement of the glycogenic function the levulose tolerance test has been applied. This test is based on the theory that levulose is rapidly absorbed from the digestive tract in the normal individual and is very easily formed into glycogen by the liver. During the whole process, no marked rise of blood sugar occurs. In liver disease, glycogen formation is interfered with, and the subsequent rise of blood sugar is due to levulose which has not been stored or utilized. The present interest in levulose tolerance is due largely to the work of McLean and Wesselow, who noted that levulose caused only a small rise in the blood sugar of normals. Modern animal experimentation has been done by Bodansky, using three grams of levulose per kilogram of body weight. No normal animals showed a rise in blood sugar greater than 35 mgm. after the administration of the levulose. In dogs, after prolonged chloroform anesthesia, a sharp drop in levulose tolerance was found, due presumably to liver injury. The presence of actual liver damage was proven by necropsy. In animals which recovered, the levulose tolerance improved as liver regeneration occurred.

These tests as applied in the average case of obstructive jaundice have not given definite results, as the changes according to these criteria are not marked. The measuring of functional changes which are probably of gradual development is unsatisfactory because of the marked ability of regeneration, as mentioned above, and because of the fact that the different hepatic functions are probably more or less dissociated, and an impairment in one, such as a levulose tolerance, does not necessarily mean an impairment in others. As to the liver function, it may be more accurate to classify these patients with obstructive jaundice as suffering from a lack of liver function rather than from hepatic insufficiency. The condition is probably one not so much a lack of vigor of the liver

cell, as the inability to function properly against an obstructed avenue of excretion. It is certain, however, that these cases present a central atrophy associated with a cholangitis, a condition which unchecked and unaided may lead to fatal termination from a lack of hepatic function.

The clinical picture presented by this complication is definite: the first week of the post-operative course may be uneventful. The biliary drainage, which up to this point may have been normal, becomes much more profuse. With this increase in flow, there is a marked thinning of the bile with gradual loss of color. The profuse drainage of bile subsides within twenty-four to forty-eight hours, but the bile remains thin. Collapse gradually appears, the pulse becomes small, and the temperature subnormal. Toxic symptoms appear—restlessness and irritability, which gradually give way to somnolence, followed by coma, and death gradually ensues. With the development of these symptoms the jaundice does not deepen, but beneath the jaundice discoloration is an increasing pallor.

Realizing the lack of hepatic function in cases of obstructive jaundice, it is necessary that we bear in mind the functions of the liver, in outlining a procedure to counteract this lack. In view of the role played by the liver in carbohydrate metabolsm, it is surprising that these patients rarely show glycosuria. Opie and Alford[3] found that on giving carbohydrates, fats, and proteins to dogs, before and after chloroform poisoning, that the dogs fed with fat died first, those with protein died second, and those fed with carbohydrate died third or not at all. From this he conclues that carbohydrates act as a protective agent to prevent the disintegration of body proteins when the individual is in a state of toxemia. By intravenous, subcutaneous, and intraduodenal injections of glucose, Mann and Magath[4] have been able to keep dogs alive twenty to thirty-four hours after removal of the liver. In view of observations such as these, jaundiced patients have been given large quantities of carbohydrates. Glucose intake has been increased by the rectal administration of 15% solution of glucose by means of the Murphy drip, administered intermittently for one hour every other hour. The eating of reasonable amounts of candy should be encouraged. (The administration of insulin has been suggested as an aid in the carbohydrate metabolism.)

When bile enters the circulation, the injury is not restricted to the liver alone. The toxic process, however complex it may be, gives rise to many combinations of symptoms. The combination of urinary changes and jaundice is not uncommon. That a toxic nephritis may develop in the course of an obstructive jaundice has long been recognized and believed to be a result of the cytotoxic action of bile salts. A large proportion of patients with obstructive jaundice show definite albuminuria and the presence of casts. The blood urea may be accepted as the definite criterion of the renal condition as applied to the advisability of operative procedure. In the majority of these cases there is not the degree of impairment of hepatic function that a low blood urea might be interpreted as a result of an hepatic insufficiency. Beyond the pre-operative recognition of this complication, its earliest appearance as a post-operative complication must be observed. Its symptomatic development can be definitely differentiated from that of hepatic failure. Although the post-operative obstructive jaundice case commonly shows a marked increase on the blood urea content, more so after general than local anesthesia, this should return to the pre-operative level within three or four days. The first symptom accompanying its continuance is a marked and sudden cessation of the drainage of bile, accompanied by a steadily increasing albuminuria and cylindruria. The jaundice gradually deepens, and the coagulation time increases. During this period of renal insufficiency secondary to the increasing jaundice, a progressive elevation of the blood urea level occurs, and the patient proceeds into a condition of uremia. The treatment is then that of uremia, with special observation of drains to see that there is no external mechanical obstruction of the biliary drainage. It is interesting to note in this connection that Cumston[5], in a review of the conclusions of many French observers, points out the fact that icterus may accompany acute nephritis with little or no manifestations in the liver. The retention of nitrogenous bodies in the blood is thought to be due to a renal insufficiency and to hyperfunction of the liver. So that the lowering of a high blood urea in jaundice may be due to an improved renal elimination or to a lowering of hepatic function. In this connection a pre-operative estimation of the renal efficiency seems important.

By far the most frequent post-operative complication of obstructive jaundice is secondary hemorrhage. The tendency of these patients to post-operative bleeding has long been recognized. In the absence of exact criteria, many indefinite clinical signs have been relied on as the index to the probability of this complication. To some individuals subcutaneous hemorrhagic spots on the patient with obstructive jaundice serve as a warning of a fatality in case of operation; others regard a coagulation time of more than nine minutes or a dehydration associated with a jaundice of more than two or three weeks' duration as definite contraindications to operation. For this reason it is probable that many cases of obstructive jaundice with a considerable question as to the type of obstruction—whether stone or malignancy—

have been denied the opportunity of an exploratory procedure.

Necropsy findings in operated cases of obstructive jaundice have shown the cause of death in more than fifty per cent of these cases to be post-operative intra-abdominal hemorrhage. The delay in the coagulation time of the blood in this condition has been the subject of much experimental investigation. Morowitz and Bierich[6] found that the bile acids are not at fault, because even in the most severe cases, the blood does not contain sufficient cholates to account for the delay. A diminished fibrin content could not be demonstrated. It has been suggested as due to a delayed formation of the fibrin enzyme, probably on account of a deficiency of thrombokinase, but the reason is not clear. That icterus is accompanied by absorption of substances liberated in the destruction of liver cells is an unproved hypothesis.

The changes responsible for this hemorrhagic tendency are based fundamentally on the toxicity of bile. Pigmentation of the tissues of the body in jaundice depends on the presence in them of bile pigments, which have been reabsorbed through the lymphatics. Present with the abnormally large quantity of bile pigment are also other less important constituents of the bile, normally not present in the systemic circulation. Bunting and Brown have shown that whole bile of rabbits is fatal to rabbits on intravenous injection of 0.12 to 0.25 c.c. per kilogram of body weight. The relative toxicity of the bile pigments and the bile salts and their respective role in the production of blood changes is still an undecided question.

By many workers it has been claimed that the bile pigments are much more toxic than the bile salts, and by an equal number this observation has been contradicted. Bilirubin is normally present in the blood, and always increased in any degree of icterus. Van den Bergh estimates bilirubin in normal blood serum as 1 part in 50,000, and when this combined pigment is more toxic than in combination with calcium or magnesium; bile from which the pigment is removed has very little toxicity. These observations may be carried further to the logical suggestion that the available calcium of the human organism is consumed in combining with the circulating bile pigments as a protective, detoxifying mechanism, leaving insufficient calcium for the normal process of blood coagulation. The decrease in available calcium may be responsible for the bradycardia and some of the mental and nervous symptoms. The bradycardia is explained by the holders of the "bile salt toxicity theory" on the vagus stimulating effect of bile salts, which has been prevented by the administration of atropine during experimentally produced obstructive jaundice. The hemolytic property of bile salts, with a resulting increase formation of pile pigments to produce more liver duct obstruction may establish a vicious circle in the progress of the condition.

Whether we accept the bile pigment or the bile salts as the toxic agent, the lack of available calcium in this condition seems definite.

Contrary to the observations of some, we have found no decrease in the level of blood calcium in jaundiced animals, as previously reported[7]. The blood calcium content determined by the Rimmer-Tistall method in both normal and jaundiced dogs ranged from 10 to 11.5 milligrams per 100 c.c. In the experimental determination of the lethal dose of intravenous calcium chloride, an incidental but interesting observation was made. A 10% aqueous solution was administered intravenously at the rate of 1 c.c. per minute until death occurred. The calcium content of blood withdrawn at the time of death was approximately equal in both normal and jaundiced animals, yet the amount of calcium injection required to effect death in the jaundiced animal was double that taken by the normal, unjaundiced dog. The readings are shown in the following table:

SUMMARY OF RESULTS OF LETHAL INJECTION OF CALCIUM CHLORID

		Weight kg.	Lethal dose mg. for each kg.	Times the therapeutic dose	Blood calcium Before	Blood calcium After	Mg. of calcium chlorid for each mg. increase in blood calcium
Normal dogs	G267	6.8	225	27.27	11.5	36.5	9.0
	2B	10.7	287.85	34.89	10.3	42.9	8.82
Jaundiced dogs	G261	7.5	304	36.8	10.3	26.9	18.31
	G166	7.8	469.23	56.8	10.9	38.7	16.87

tent is exceeded the pigment is deposited in the skin and excreted in the urine. In the work of King and Stewart[2], the following observations were made: the amount of pigment in a lethal dose of whole bile will cause death, but the bile salts present in the same amount of bile will produce no demonstrable effects; uncom-

The figures in the last column of the tabulation were obtained by dividing the number of milligrams of calcium chlorid taken for each kilogram of body weight by the number of milligrams for each 100 c.c. increase in the blood calcium content. These figures, showing that it requires approximately double the amount of

injected calcium to raise the blood serum calcium content of the jaundiced dog to the same level as that of the normal dog, in spite of the fact that the blood calcium content is practically the same before and after a lethal dose, suggests a lack of free, available calcium ions in the jaundiced animals which is not apparent in the blood serum calcium taken before injection.

The definite fact remaining is the tendency of the jaundiced patient to post-operative secondary hemorrhage and our ability to estimate that tendency according to the coagulation time of the blood of the individual patient.

There are available two simple procedures for determining the coagulation time: Bogg's Method, which consists in noting the time required for the coagulation of the blood flowing from a stab wound in the ear; and the method of Lee and White, noting the time required for the coagulation of one cubic centimeter of blood in a clean tube and obtained by venupuncture with a syringe, both the tube and syringe having been previously washed with normal saline solution. The latter method is to be preferred, because in the Bogg's procedure true coagulation may be abnormally hastened by contamination with the tissue juices liberated in the incision of the lobe of the ear. In conjunction with the coagulation time, the so-called "calcium time" of the blood is important and corroborative of the deficiency of available calcium. This may be determined by the procedure described by Lee and Vincent: six drops of a 0.5 per cent solution of calcium are added to 1 c.c. of venous blood, and the time required for the coagulation of this mixture is called the calcium time of the blood. When the calcium time is shorter than the coagulation time of the blood, the coagulation time can be lowered through the administration of calcium.

Both oral and intravenous administration of calcium have been carried out. Calcium lactate, 100 grains per day for three days, has given satisfactory results in some quarters. Grove and Vines[8] have reported experimental results from which they conclude that calcium salts given by mouth have no influence on the blood calcium. Because of these reports and the low alimentary absorption of calcium, the intravenous method seems preferable particularly in view of the fact that a considerable portion of the administered calcium is, theoretically at least, to combine with and detoxify the circulating bile pigments, the excess serving to restore the blood calcium to a normal level.

Calcium chlorid has been adopted as the preparation for this use, and is given as a sterile 10% aqueous solution, in doses of 5 c.c. intravenously for three successive days. At the end of this period, the coagulation time has ordinarily been reduced to normal, and will be held there for five to seven days, at which time the procedure may be repeated if indicated.

The intravenous administration of calcium has naturally given rise to the question of its toxicity. Cushing states that "calcium injected into the blood stream acts much like digitalis in that in small doses it accelerates and strengthens the heart, but in large doses it seems to be poisonous, tending to bring the heart to a stand-still." We have previously reported[9] work on the electrocardiographic effects of intravenous calcium. The injection of 10% solution of calcium chlorid into the blood stream of a dog will produce various alterations in rate, disturbances of conduction, ectopic origins of impulse, and when carried to massive lethal doses disturbance of the fundamental coördination of the heart and ventricular fibrillation, which is followed by the death of the animal. Therapeutic doses have produced only alterations of rate, of which the patient will be conscious during the injection. In administering calcium intravenously to patients, one is impressed with the usual quickening of the pulse rate and the sensation of flushing of heat over the entire body, which are very temporary effects to which Hirschsohn and Maendl[10] give the name of "dynamic effect" of intravenous administration. The same phenomenon was observed by them following the injection of quinine, dextrose, and urea. For this reason the intravenous administration should be carried out very slowly, taking three to five minutes for the injection of the 5 c.c.

The elimination, or rather the disappearance of injected calcium chlorid from the blood stream is rapid, removing any danger of accumulation of successive doses. With the therapeutic doses suggested, the rise in blood calcium is immediate, and a return to normal blood calcium level occurs within two hours[8]. The fate of this calcium is not known, though because of the small amount excreted it probably undergoes an obscure storage process. As previously stated, the coagulation time will usually be reduced to normal after three daily injections. At that time, on the day preceding operation coagulation tests may be repeated, and further calcium administered if necessary. A coagulation time of nine minutes has been adopted as the upper limit of safety preceding surgery. In the very infrequent cases failing to respond fully to the administration of calcium, transfusions may be used to accomplish a satisfactory lowering of the coagulation time.

Lastly, it must be borne in mind that obstructive jaundice of any appreciable duration is frequently accompanied by infection, to which a part of the intoxication is undoubtedly due. For this reason a large fluid intake is encouraged, and routinely it is adjusted that these cases receive 4000 c.c. of fluids per twenty-four hours during the period of preparation. This period of preparation ordinarily lasts from three to four days, when the coagulation time should have been reduced practically to

normal, the general condition of the patient accurately estimated, and the surgeon presented with a much more satisfactory surgical risk.

SUMMARY

The patient with obstructive jaundice presents an increased surgical risk. The common post-operative complications are a lack of liver function, uremia, and post-operative hemorrhage. Pathologically, these are based on a biliary toxemia usually accompanied by infection, resulting in a toxic hepatitis and nephritis, and by a lack of available calcium. The surgical risk in these cases can be markedly reduced by proper pre-operative management.

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STUDIES IN LIVER FUNCTION. III. METHODS FOR DETERMINING THE FURFUROL NUMBER AND THE BILIRUBIN CONCENTRATION OF DUODENAL CONTENTS*

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THE present communication describes a method for estimating the concentration of the bilirubin found in duodenal contents, and also a simplification of the bile acid method previously reported. These methods are additions to a series of methods which have been published[1], [2]†. All of them represent arbitrary procedures for estimating the concentrations of various components of the bile present in duodenal contents. By determining the normal concentrations of these components a basis is afforded for ascertaining how they are influenced by diseases affecting the liver. In this way data are obtained which bear on the functional condition of the liver.

METHOD FOR DETERMINING THE "FURFUROL NUMBER"

This method is a modification of the previously described procedure for determining the bile acid concentration (2) of duodenal contents.

Reagents. The reagents necessary are: Merck's furfurol (reagent), which is a chemically pure preparation of furfurol; chemically pure sulphuric acid and Schuchardt's preparation of glycocholic acid.

(a) Three-tenths (0.3) per cent furfurol solution. This is prepared by dissolving three (3) cc. of furfurol in about 700 cc. of H_2O in a litre volumetric flask, and making up to the mark with H_2O. (b) Fifty (50) per cent volume H_2SO_4. This is prepared by mixing equal volumes of H_2SO_4, (sp. gr. 1.84) and H_2O, and is allowed to cool before using.

(c) Glycocholic acid standard. This is prepared by dissolving 100 mgms. of glycocholic acid in about 30 cc. of N/10 NaOH solution in a 50 cc. volumetric flask and making up to the mark with the same solution.

Method. Add one (1) cc. of duodenal contents to nine (9) cc. of 95 per cent alcohol in a test tube and mix thoroughly. Filter through a dry filter paper. Add one (1) cc. of the clear filtrate to a test tube containing six (6) cc. of the 50 per cent H_2SO_4, and one (1) cc. of the 0.3 per cent furfurol solution and one (1) cc. of H_2O. The whole is thoroughly mixed by rotating the tube. After this mixing the test tube is placed in a water bath at 67 degrees C for thirty (30) minutes. At the end of this period the tube is removed from the water bath and cooled by placing in tap water. When necessary the colored solution may be clarified by centrifugalization. A mild degree of turbidity does not make sufficient error to vitiate the clinical value of the result.

The glycocholic acid standard is prepared by adding one (1) cc. of the standard solution to the mixture of 6 cc. of the 50 per cent H_2SO_4, and one (1) cc. of 0.3 per cent furfurol solution and one (1) cc. of 95 per cent alcohol. This is heated in the water bath along with the unknown. Color comparisons are made in the Hellige colorimeter, using the scale reading from below upwards. The reading of the unknown on this scale is arbitrarily multiplied by two; and the resultant figure is expressed as the "furfurol number."

METHOD FOR THE QUANTITATION OF THE CONCENTRATION OF BILIRUBIN OF DUODENAL CONTENTS

The present communication outlines a method for the quantitative estimation of the concentration of a brownish colored pigment found normally in duodenal contents obtained after stimulation of the flow of bile by the lavage of the duodenum with 33 per cent magnesium sulphate

*From the Gastroenterological Laboratory of the Department of Biochemistry, Evans Memorial, Boston.
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solution or with oleic acid. For purposes of description this pigment, or mixture of pigments, has been given the designation, bilirubin.

METHOD

The chemicals required should be chemically pure unless otherwise designated. They are as follows: Calcium hydrate; sulphanilic acid; sodium nitrite; chloroform, U. S. P.; 95% grain alcohol, U. S. P.; bilirubin (biliphain)* (obtained from Eimer and Amend, New York City). The apparatus required is as follows: test tubes; 15 cc. capacity centrifuge tubes; 5 cm. diameter glass filter funnel with ordinary filter paper to fit. One, three, five and ten cc. volumetric pipettes. A Duboseq colorimeter. A centrifuge.

PROCEDURE

Reagents: A 10 per cent suspension of calcium hydrate in distilled water. 0.5 per cent sulphanilic acid solution is prepared by placing 5 gms. of sulphanilic acid in a litre volumetric flask, adding about 800 cc. of distilled water, dissolving and making up to the mark with distilled water; then adding a few cc. of chloroform to prevent the growth of fungi. This solution is stabil.

0.02 per cent solution of sodium nitrite is prepared by placing 0.1 gm. of sodium nitrite in a 500 cc. volumetric flask, dissolving in distilled water and making up to the mark with distilled water.

0.2 per cent solution of biliphain is made by placing 10 mgms. of biliphain in a 50 cc. volumetric flask, dissolving in chloroform and making up to the mark with chloroform. It is not known just how long such a solution will keep, but it is good for use for fully two months.

HCl solution: this is prepared by adding 25 cc. of the usual chemically pure solution of HCl to 75 cc. of distilled water.

$MgSO_4$ solution is prepared by dissolving 25 grams of crystalline $MgSO_4$ in 100 cc. of distilled water.

The usual 95 per cent U. S. P. grain alcohol must be redistilled. This is best carried out on an electric hot plate.

PROCEDURE

Pipette 10 cc. of duodenal contents, if yellow, 5 cc. if brown in color, into a 15 cc. centrifuge tube, add 1 cc. of $MgSO_4$ solution and then 3 cc. of the 10 per cent calcium hydrate suspension and mix with a glass rod; wash off the rod with water and centrifuge until clear. Decant the clear and colorless supernatant fluid. Add 5 cc. distilled water to the precipitate, mix with a glass rod, add 5 cc. of a mixture of one part of HCl and three parts of distilled water, and mix until the calcium hydrate has all dissolved. Then wash off the rod with water and centrifuge until the supernatant fluid is opalescent. This fluid should not give a color reaction with the sulphanilic acid-nitrite mixture. Again decant, add 10 cc. of alcohol, mix the precipitate with the alcohol, grinding up the flocculent particles, and again centrifuge until the supernatant fluid is clear. Repeat this process of washing with alcohol three times. The alcohol from the third washing should contain no more than a trace of bilirubin. After the third washing, decant the alcohol completely, and add 3 cc. of chloroform and mix with a rod. Then filter through the small filter paper. Add one (1) cc. of this chloroform solution to the following mixture in a test tube, made in exactly the order given: one cc. of the 0.5 per cent sulphanilic acid solution, one cc. of the 0.2 per cent sodium nitrite solution, 4 cc. of the alcohol. Mix the contents of the tube by rotation and allow to stand for thirty minutes. Then read the color which develops against the color developed by one cc. of the standard biliphain solution prepared in the manner as described, and at the same time as the unknown. For color comparison use the Duboseq colorimeter.

The index of bilirubin concentration is expressed in mgms. per 100 cc. of duodenal contents.

DISCUSSION

It must be recognized frankly that the chemistry of both the so called bile acids and bile pigments is in a most unsettled state. This renders the reliability of any method for their quantitation uncertain. For this reason the term "bile acid concentration" has been changed to that of "furfurol number"; and, also, that of pigment concentration expressed in per centum will be changed to "pigment concentration number" in future communications. In spite of the unsettled state of the chemistry of the substances determined, the methods described here give uniform results. They have been found useful in a procedure for estimating the state of liver function, which is the reason for publishing them.

The method described for obtaining the furfurol number entails a simple technical procedure. However, the shades of color of the standard and of the unknown are not exactly similar. Nevertheless, experience has shown that this difference neither interferes with nor renders difficult the accurate comparison of the two intensities of color in the colorimeter. Observations on pathological duodenal contents have demonstrated that variations in the furfurol number may occur independently of those in the concentrations of cholesterol, total pigments or bilirubin. Therefore, the furfurol number represents an independent entity of duodenal contents.

It is generally accepted that the pigment

found in normal bile is bilirubin. However, in the bile fraction of duodenal contents there are at least two classes of bile pigments; as demonstrated by their relative solubilities in alcohol. For purposes of description the pigment which is relatively insoluble in alcohol is called bilirubin. The concentration of this so called bilirubin varies directly with the depth of brown color shown by the duodenal contents. Similar to the furfurol number, the concentration of bilirubin has been found to vary independently of the other constituents of the bile fraction of duodenal contents. Therefore, "bilirubin" represents another independent entity present in the bile fraction of normal duodenal contents.

The clinical significance of the furfurol number and bilirubin concentration will be discussed in a subsequent communication.

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Studies in Liver Function. IV. A Procedure For the Uniform Stimulation of the Biliary Flow*

BY C. W. MC CLURE, M.D., W. L. MENDENHALL, M.D., AND M. E. HUNTSINGER

THE present communication reports results obtained from the continuation of investigations on liver function, as estimated by the study of the biliary elements of duodenal contents. Methods for the uniform stimulation of a flow of bile and the findings from it will be described. These findings will include (a) the gross color, (b) the furfurol number[1], (c) the total pigments[1], concentrations of (d) bilirubin[1], and (e) cholesterol[2] of the duodenal contents obtained from both normal and pathologic subjects.

In an investigation previously reported[3] the estimation of the functional condition of the liver through the study of the bile fraction of duodenal contents was discussed. The results presented indicated that this method for ascertaining the level of liver function promised to be more delicate than any previously proposed. However, during the investigation it was found that magnesium sulphate solution, which was used to stimulate the flow of bile, occasionally failed to do so in the normal subject. The explanation for this failure is offered by observations[4] on animals; which show that the improper use of magnesium sulphate leads to depression of the biliary function of the liver. This finding makes it difficult to decide whether a given abnormal duodenal bile has resulted from lack of stimulation of the liver by magnesium sulphate or from a functional disturbance of that organ. For this reason, experimentation was continued until substances were found which would stimulate uniformly the excretion of bile in both normal persons and animals. Of these substances, oleic acid proved to be the most suitable for the purpose of the investigation reported here.

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PROCEDURES

For the purpose of the present investigation subjects were given the duodenal tube with the stomach in a fasting state. The metal tip of the tube was allowed to enter the second portion of the duodenum, where its position was verified by fluroscopy. The subject then reclined on the right side and a mixture of five (5) cc. of oleic acid and 45 cc. of warm tap water were poured through the tube into the duodenum. The proximal end of the tube was closed by a clip for fifteen (15) minutes. At the end of that period, collection by siphonage of duodenal contents was begun. The first sample or fraction collected contains oleic acid and frequently gushes out the end of the tube. Experiment has demonstrated that this portion is to be discarded. Collection for analytical purposes is then begun, and continued for thirty (30) minutes. However, if at any time during this thirty-minute period the material as it flows suddenly exhibits a marked change in color, a new collecting flask is substituted and the differently colored contents are collected for the ensuing thirty (30) minutes. This method of procedure allows for variable rates in absorption from the intestines; and for possible differences in the latent period of stimulation of the normal liver.

The gross color of the duodenal contents was judged directly by inspection. The analyses for cholesterol[2], pigments[1], [5] and furfurol number[1] were carried out by the methods already described.

The duodenal contents of twelve apparently normal subjects were collected and examined according to the procedure outlined above. The subjects ranged in age from 23 to 50, and included both sexes. The results obtained from this study are collected in Table 1 which follows.

A striking feature of the results outlined in Table 1 is that all the specimens of duodenal contents were definitely brown in color. Another feature to be noted is that the quantity of each constituent tends to vary independently; i. e., the contents of Subject No. 12 contained 50 mgms. of cholesterol and 17.4 mgms. of bilirubin, while those of Subject No. 2 contained more than three times that amount of cholesterol but only 0.8 mgm. more of bilirubin. In addi-

tion to the study outlined in Table No. 1 duodenal contents were studied from six normal subjects after stimulation of the flow of bile with magnesium sulphate solution. The results paralleled those derived after the use of oleic acid as a biliary stimulant. This parallelism was so similar that no further description of the results seems necessary.

In a former communication[3] it was found that the examination of duodenal contents obtained after lavage with magnesium sulphate solution afforded decided evidence of disturbance in hepatic function in the presence of lesions affecting the parenchyma, circulation or ductal system of the liver. Also, as mentioned above, it has been found that when oleic acid is used to stimulate the normal liver it gives rise to a bile comparable in its constitution to that obtained when magnesium sulphate was the agent of excitation. On the basis of these findings, obviously the practical value of the use of oleic acid as a biliary stimulant rests on whether it will give rise to the excretion of a type of bile comparable to that obtained from the duodenum of pathologic persons after the use of magnesium sulphate solution. To ascertain the comparative effects of these two substances, duodenal contents were collected from patients after introducing oleic acid into the duodenum, and the results obtained compared with those from patients with the same type of pathological condition after using magnesium sulphate solution as a biliary stimulant. The conditions included hepatic cirrhosis, passive congestion of the liver and acute or chronic cholecystitis; and, also as controls, patients without known biliary pathology. The results from the use of oleic acid paralleled those obtained from the use of magnesium sulphate solution[4]. Since the latter have been described elsewhere, no elaborate discussion of those derived from the use of oleic acid seems necessary.

Table 1.

Character and analyses of the biliary fraction of duodenal contents from normal subjects after the use of oleic acid.

Subject	Pigment concentration number	Bilirubin concentration in mgms. per 100 cc.	Furfurol number	Cholesterol concentration in mgms. per 100 cc.	Color	Amount in c.c
1	300	16.0	150	137	brown	60
2	500	18.2	212	167	brown	70
3	500	19.0	130	213	brown	30
4	250	22.5	140	58	brown	50
5	114	8.4	100	32	brown	50
6	100	7.1	100	52	brown	100
7	142	6.7	142	30	brown	50
8	115	8.7	110	75	brown	60
9	350	13.5	116	52	brown	60
10	576	9.1	220	100	brown	40
11	475	16.8	110	60	brown	70
12	390	17.4	94	50	brown	50

The following table (Table No. 2) gives a resume of the findings in normal duodenal contents and permits a comparison of them with the findings in abnormal contents, using oleic acid as the biliary stimulant.

TABLE 2

Results of analyses of the biliary fraction of duodenal contents obtained from patients.

Case No.	Cholesterol in mgms. per 100 cc. duodenal contents	Pigment concentration number	Furfurol number	Bilirubin in mgms. per 100 cc. duodenal contents	Color	Diagnosis
	50–213	100–500	94–220	6.7–23.5	Brown	Normal limits observed
1	16	43	42	0.0	Pale yellow	*Cirrhosis of liver; no jaundice
2	Trace	180	21	Trace	Dark green	Hepatic fever after excision gall bladder
3	Trace	37	Trace	Trace	Pale yellow	*Acute cholecystitis no jaundice
4	12	114	30	Trace	Pale yellow	*Chronic cholecystitis calculi, no jaundice
5	Trace	50	30	4.2	Brownish yellow	*Cirrhosis of Banti's Disease, no jaundice
6	55	1650		39.0	Reddish purple	*Chronic hemolytic jaundice; icteris
7	24	40	0.0	0.0	Pale yellow	Vascular hypertension with dyspepsia
8	17	102	74	1.8		Migrane
9	44	67	90	1.9	Yellow	Nausea, vomiting (no lesions found during exploratory laparotomy).

* Diagnosis confirmed on laparotomy.

Perusal of Table No. 2 shows that the color of normal duodenal contents is always brown in color, while that of abnormal contents may be of various colors. The concentrations of the constituents of normal contents show considerable variations; but the concentration of the constituents of abnormal contents is so widely different from the limits of normal variations as to make a sharp distinction between the two readily possible.

SUMMARY AND CONCLUSIONS

In the present investigation oleic acid has been found to be a uniform stimulant to the flow of bile in both normal man and animals. The results obtained by the study of normal human duodenal contents in this investigation after stimulation of the flow of bile by oleic acid or by magnesium sulphate solution have been found to be similar. This makes a total of eighteen subjects studied, which is considered to afford sufficient data to establish the character of normal duodenal contents obtained after the use of oleic acid. A series of animal experiments to be described elsewhere substantiate completely this conclusion. The findings show that the examination of duodenal contents as

described furnishes a fairly delicate index to the state of liver function.

The origin and metabolism of the various constituents of the bile are given added interest to the clinician by their use as guides to the state of liver function. However, so little is known about the chemistry of these substances that not much of authoritative nature may be said concerning either their origin or their metabolism. Cholesterol is usually considered as an excretory product in the bile. Bile acids are possibly formed in the liver. But the opinion formerly held, that bile pigments are formed in the liver, is now seriously questioned by biochemists; indeed, the current belief seems to be that they are in part, at least, merely excreted by that organ. However, observations made during the present investigation on patients with liver functional derangements, both those with obstructive jaundice and those without any traces of jaundice, show that both may excrete in the bile similar concentrations of an alcohol-soluble pigment but no alcohol-insoluble pigment whatsoever. In view of this finding the absence of jaundice makes it appear that the alcohol-insoluble pigment is more than merely an excretory product of the liver. Both classes of patients will show absence of the alcohol-insoluble pigment for many weeks and until the state of liver function improves; then that pigment will make its appearance in progressingly increasing concentration. Such findings suggest that this particular pigment, which in the present investigation has been arbitrarily called bilirubin, is formed in the liver; i. e., a product of its metabolism. On the other hand, in familial hemolytic jaundice both the alcohol-soluble and -insoluble pigments may be found in normal concentrations. Nevertheless, the duodenal bile may be of an altogether abnormal color. Apparently in this clinical condition an abnormal type of pigment occurs in the bile; which may theoretically be considered as arising outside the liver, a view long held byclinicians. However, the extrahepatic origin of this or of any of the bile pigments cannot be definitely settled, because there are no available means for positively identifying the presence in the blood of exactly the same types of pigments which may occur in the bile. Indeed, in the present investigation two types of bile pigment have been found in the duodenal bile, in the bile removed from a human gall bladder, and in the bile from both dogs and cats[4].

Biochemists working on so called bilirubin in the blood do not seem to have considered the possibility that bile may contain a complex of pigments. It is theoretically possible that what biochemists are considering as bilirubin in the blood may be only precursors of bile pigment derived from hemoglobin; or, a pigment complex resulting from the toxic conditions arising from the character of the experimental conditions; or, indeed, it is possible that it is an abnormal type of pigment arising in hemolytic jaundice. For these reasons and because of the vast importance to the clinician of a proper conception of the true functions of the liver, it might be wise for those of us who are practising medicine to withhold judgment on the merits of the present controversy as to the origin of the normal bile pigments until more definite knowledge concerning them is acquired.

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Studies in Liver Function. V. Clinical Observations on the Evaluation and Treatment of Disturbed Liver Functions*

BY C. W. MC CLURE, M.D., M. E. HUNTSINGER, B.S., AND J. GOTTLIEB, M.D.

THE present communication outlines the principal results obtained from a clinical investigation of the evaluation and treatment of disturbed liver function. The investigation includes two studies. The first study is concerned with the relation between the symptomatology of patients with gastrointestinal disturbances and the state of liver function. The second study comprises the equation between the status of the patient's condition and the coincident state of liver function. The functional condition of the liver was evaluated by examination of the biliary elements of the duodenal contennts, following procedures already reported[1]. Sufficient numbers of observations have been made by these procedures to show that the results obtained from them afford a fairly delicate index to the condition of hepatic function.

The treatment of disturbed liver function consisted of the introduction of magnesium sulphate solution into the duodenum through a tube; as was first proposed by Lyon[2]. Until recently about the only criterion available for judging the therapeutic value of that procedure has been that offered by changes in the clinical manifestations. However, it is generally agreed that such changes can be the result of factors other than the action of magnesium sulphate. For this reason the therapeutic value of the procedure has been the subject of much controversy. Very excellent articles presenting the favorable and unfavorable aspects of this controversy, together with collections of the literature, are those of Soper[3] and of Jones[4]. Obviously, the therapeutic effect of placing magnesium sul-

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phate solution in the duodenum can only be adequately investigated by the use of methods which will give direct objective evidence of any action it might produce. Such objective evidence is afforded by examination of the bile fraction of duodenal contents according to the procedures already reported. One reported study[5], using some of these procedures, gave results indicating that magnesium sulphate influenced the functional activity of the liver. The development of more refined procedures[6] has permitted an amplification of that study, and the results are discussed in the present communication.

For purposes of the present investigation patients were given the duodenal tube and the flow of bile established through the use of oleic acid according to the procedures described in a previous communication[1]. The duodenal contents were examined for their gross color, the concentrations of cholesterol, total pigment and bilirubin and for the furfurol number by the methods described in that communication. The procedure of case study comprised the history, physical examination, the usual clinical examinations of blood, urine and stools, the Wassermann reaction on the blood serum, and X-ray studies of the gastrointestinal tract. The types of cases studied were those of acute and chronic cholecystitis, cirrhosis of the liver, peptic ulcer, a group with gastrointestinal symptoms of uncertain etiology, and migraine.

Out of a much larger number of patients, in whom the clinical diagnosis of chronic cholecystitis was made, twenty were selected whose findings will be reported. These patients have been selected for the following reasons: (1) Their symptomatology was typical. (2) The bile fraction of the duodenal contents of all of them was initially abnormal; i. e., liver function was deranged. (3) The character of the symptomatology was such as to make it seem that any relief afforded would be more than a mere coincidence. (4) The patients could be followed long enough to give a fair conception of the immediate results of the intraduodenal therapy used. The initial findings in the duodenal contents, those representative of the effects of treatment, the length of the period of treatment, the final result of the progress of the case and the period of observation are outlined in Table No. 1, which follows:

TABLE 1.

The effects of intraduodenal therapy on the state of liver function and the symptoms of patients diagnosed as chronic cholecystitis.

Case no.	Findings in duodenal contents before and after treatment										Effect of therapy on liver function	Effect of therapy on symptoms	Number of weeks of initial treatment	Number of months the patient observed after completion of initial treatment
	Pigment concentration number		Bilirubin concentration		Furfurol number		Cholesterol concentration		Color					
	before	after	before	after	before	after	before	after	before	after				
1	38	45	3.6	7.2	50	80	16	28	yellow	brown	improved	improved	30	12
2	87	276	0.0	13.2	40	166	17.6	77	yellow	brown	normal	relieved	15	14
3	32	260	0.0	7.2	trace	178	trace	63	yellow	brown	improved	relieved	8	5
4	60	132	0.0	0.0	trace	62	13.5	21	yellow	yellow	improved	relieved	6	3
5	32	85	0.0	4.1	30	48	14	20	yellow	brown yellow	improved	relieved	8	2
6	90	240	3.6	4.7	32	58	14.4	35	yellow	brown yellow	improved	relieved	8	2
7	80	80	2.2	4.5	60	74	16	56	yellow	brown yellow	improved	relieved	28	12
8	65	111	trace	6.0	20	70	trace	42	yellow	brown	improved	relieved	9	8
9	207	140	0.0	11.4	70	78	26.9	42	yellow	brown	improved	relieved	8	5
10	16	124	0.0	8.2	52	150	13	27.5	yellow	brown	improved	improved	11	2
11	55	72	0.0	8.9	42	34	20	21	yellow	brown	improved slightly	improved slightly	18	2
12	51	175	0.0	7.8	30	100	12	46.4	yellow	brown	normal	relieved	10	2
13	78	76	0.0	8.2	40	120	12.3	30.3	yellow	brown	improved	relieved	8	3
14	126	190	trace	16.0	24	60	20	46.5	yellow	brown	improved	relieved	9	5
15	40	325	3.0	16.4	34	162	14	80	brown yellow	brown	normal	relieved	7	6
16	18	52	0.0	3.2	trace	44	trace	22	yellow	brown yellow	improved	relieved	9	8
17	65	100	0.0	4.9	trace	15	16	28	yellow	brown yellow	improved	improved	15	6
18	105	100	3.0	3.0	34	40	trace	27	yellow	yellow	improved slightly	improved moderately	19	4
19	40	168	0.0	9.3	22	86	14	33	yellow	brown	improved	relieved	10	80
20	77	110	0.0	0.0	20	20	18.4	trace	yellow	yellow	unimproved	unimproved	8	8
Minimum normal	100		7.0		100		30.0		brown					

Study of Table No. 1 shows that amelioration of symptoms was associated with evidences in the duodenal bile of improvement in the state of liver function. Observations on the duodenal bile showed that after the patients' symptoms had disappeared repeated examinations usually showed a normal, or nearly normal, state of liver function. However, a group of patients was found whose reaction of the duodenal bile to treatment showed peculiar differences from those discussed in Table No. 1. One section of this group was relieved from symptoms but

there was little associated improvement in the duodenal bile findings. However, three months after stopping treatment the bile from the patients of this section was found to be normal, or nearly normal, on repeated examinations. The other section of the group improved but little symptomatically during the period of therapy, while the findings in the duodenal bile varied from abnormal to normal without demonstrable relation to the symptoms. Three months later some of these patients were free from symptoms and the duodenal bile was persistently normal on repeated examinations; while the same peculiar variations remained in the duodenal bile of those patients who did not improve symptomatically. The diagnosis of cholelithiasis was made on one of these patients by the finding of gall stones in the stools on three occasions. This patient was finally relieved of all symptoms and the duodenal bile became persistently normal on repeated examinations.

Regarding the merits of the therapy used in the cases discussed, it seems that the results warrant the statement that during the period of therapy a relatively considerable number of patients, with apparently chronic cholecystitis, improved definitely. Therefore, the results were satisfactory enough to justify the method of treatment.

A small group of patients were studied from whom the gall bladder had been excised for cholecystitis, with or without stones, and who had suffered a return of symptoms similar to those occurring prior to operation. In two of these patients the state of liver function was found to be nearly normal; and on laparotomy extrahepatic lesions were demonstrated to be the cause of the symptoms. Two of the patients showed gall bladders bulging with stones; because of which total excision was performed. Following this both patients suffered far more from biliary colic than before the operation. Such pains had persisted daily for six weeks in one patient and three months in the other before beginning the use of intraduodenal therapy. Within two weeks after the use of that type of treatment both patients were almost free from pain and dyspepsia. One of these patients was treated for six months and one for five weeks; following this they have remained apparently well for six months and for three months, respectively. Following excision of the gall bladder, another patient developed attacks of biliary colic, nausea, vomiting and fever. Such attacks occurred every few to eight months. They were not stopped by intraduodenal treatment, and the state of liver function remained very abnormal at all times. Subsequent operation showed a stone high up in the hepatic duct.

A fourth patient was relieved of all symptoms for three years following excision of the gall bladder. There then developed attacks of hepatic fever. Over a period of eighteeen months such attacks increased in frequency and severity until they became almost incapacitating. This patient was given weekly intraduodenal treatments over a period of four months. During the first four weeks of treatment all symptoms disappeared and the patient began to gain in weight and strength. The state of liver function gradually improved and at the end of four months was essentially normal. The patient has remained well for seven months after stopping therapy.

The interesting findings in this group of patients are three:—First, hepatic function was nearly normal in two and at laparotomy extrahepatic lesions accounted for the symptoms. Second, one patient showed no improvement in liver function and the symptoms were not relieved. Third, three patients were relieved of symptoms and in these patients there were varying degrees of improvement in liver function. In one of them the improvement in liver function was marked; in another it was of moderate degree; and in a third it was slight. However, in this last patient the functional state of the liver has not yet been followed long enough to establish what it may ultimately become.

Two subjects were studied whose livers were cirrhotic; the diagnosis was confirmed pathologically. One of them was not jaundiced while the other was slightly so. The findings in the duodenal bile of both were nearly the same, and showed derangement of the functional state of the liver.

Ten patients were studied in whom the findings did not warrant a positive clinical diagnosis. Two of the patients had been operated on for supposed chronic cholecystitis and no intraabdominal lesions found. Another had been diagnosed and treated as a possible duodenal ulcer. The others had been diagnosed as gastrointestinal neuroses. The histories presented by these patients are of frequent occurrence, and for this reason their salient features will be mentioned. One of the patients was a druggist of 32. For six years he had had periodic attacks of nausea and vomiting. The vomiting was preceded by epigastric distress, but pain never occurred. Vomiting occurred without relation to the time of food ingestion. Attacks persisted from two to four weeks and developed once or twice a year. The bowels were constipated, the appetite fairly good. On fluoroscopy the stomach showed an exaggerated type of peristaltic wave. Examination of the duodenal contents showed a decidedly abnormal type of bile; i. e., disturbed liver function. Intraduodenal treatment was followed by relief from symptoms and return of the bile to normal. The patient has remained apparently well for eighteen months. Another of the patients was 27. During the summer of 1924 he had developed moderately severe heartburn at irregular intervals after food. There were associated headaches and malaise. At that time the X-ray studies showed a mild degree of pylorospasm. The patient was

treated for duodenal ulcer. But he never became entirely free from symptoms. At the end of seven months his physician asked me to go over the patient. Liver function was then found to be deranged, as estimated from examination of the duodenal bile. Under intraduodenal treatment the patient became free from symptoms and the state of liver function returned to normal. Eight months later the patient remained well. Another patient was a young woman of 25. For about eight months she had suffered with nausea, during the first few months of which vomiting had occurred frequently but had largely disappeared at the time of examination. On one occasion there had been an attack of severe epigastric pain. X-ray studies were negative. The bile fraction of the duodenal contents showed a marked degree of abnormality; i. e., disturbed liver function. This patient was not treated. A young man, a student, requested the privilege of taking the tube. This was because he occasionally became nauseated and vomited. He had no other symptoms and medical studies were negative. However, the duodenal bile was decidedly abnormal, and led to the interpretation of functional liver disturbance as the basis of his vomiting. Another student had nausea and vomiting after ingesting either chicken or eggs. This student showed large cutaneous reactions to the proteins of these two foods. The duodenal bile was found to be very abnormal, and was regarded as showing the presence of disturbance in liver function. Neither of the students was treated. Two patients of this group complained of distress after food and of mild nausea and constipation. Both showed disturbances in liver function and both became free from symptoms following intraduodenal treatment. Symptomatic improvement was accompanied by improvement in the state of liver function. Another patient was a man of 50. He had had discomfort persistently present in the right upper abdominal quadrant for six weeks, and an associated loss of appetite. X-ray studies showed a decidedly abnormal type of gastric peristalsis and the duodenum never filled properly. Liver function was found to be disturbed on examination of the bile of the duodenal contents. Under intraduodenal treatment the symptoms quickly disappeared. But the patient could not be observed long enough to judge properly of the ultimate effects of the treatment on either the symptoms or the state of liver function. One patient was treated in whom nausea was the presenting symptom, and had been severe for several months. This patient improved symptomatically but little and correspondingly little change developed in the state of liver function. Thus out of a group of ten parents, seven received intraduodenal treatment. Of these, six improved definitely and apparently as the result of the treatment. Symptomatic improvement was accompanied by improvement in the state of liver function as shown by analyses of the duodenal bile.

Four patients were studied who suffered with the symptomatology typical in all respects with the clinical condition called migraine. One of the patients had, also, fairly frequent attacks resembling gall stone colic. Two of these patients showed cutaneous reactions to the proteins of foods commonly eaten by them. The state of liver function of one of these two patients was deranged, as shown by examination of the duodenal bile; while the findings in the bile of the second one were normal. The findings in the duodenal bile from the other two patients gave evidence of disturbance in hepatic function. Thus, three out of four patients with migraine showed disturbance in the state of liver function.

Twenty consecutive patients were given the duodenal tube who presented symptomatology and X-ray findings typical of duodenal ulcer. Of these, seven gave decidedly abnormal findings in the duodenal bile; and, therefore, were considered to have coexisting liver functional derangement. Two of these seven patients had been but partially relieved by apparently proper medical measures. Adding intraduodenal therapy to their treatment was followed by entire relief from symptoms and return of liver function to the conventional normal. Another patient had remained free from ulcer symptoms under diet for two years. He then developed complete anorexia and disgust for foods. Intraduodenal therapy was followed by return of appetite and taste for food; and the liver functional state changed to the conventional normal. These results suggest that the symptoms in these three patients were dependent on the functional condition of the liver. They further suggest the advisability of considering the determination of the state of liver function in ulcer patients who are not progressing satisfactorily under the usual medical methods of treatment.

SUMMARY AND DISCUSSION

The present communication has been confined to the discussion of those patients in whom the findings were interpreted as showing evidence of disturbed liver function. Because of this, it is not to be inferred that the types of clinical conditions discussed will invariably be accompanied by abnormal findings in the duodenal bile. Up to the present no attempt has been made to ascertain the relative percentages of normal and abnormal findings in the duodenal bile in these conditions. The principal reason for limiting the discussion depends on the fact that changes in the abnormal findings in the duodenal bile could be used as an index to the effects of the therapeutic measures employed. The findings in the group of patients in which the examinations of the duodenal bile were of aid in furnishing an explanation for the symptomatology have been included in the discussion because of their clinical interest.

Medical writers commonly agree that disturbances in liver function may accompany cholecystitis, and Jones[4] strongly supports such a conclusion. Thus the findings presented in this report conform to a generally accepted opinion. The same may be said of the findings in cirrhosis of the liver; for all authorities agree that in this condition the state of liver function is disturbed. The frequency of the coexistence of duodenal ulcer and chronic cholecystitis is a matter of common knowledge to all abdominal surgeons. For this reason, it is not surprising that the character of the duodenal bile gave evidence of abnormal hepatic function in approximately thirty per cent of those patients in whom the diagnosis of duodenal ulcer was made.

The patients with migraine, those showing cutaneous reactions to food proteins, and those with gastrointestinal symptoms of uncertain etiology from interesting clinical groups. The finding of an abnormal type of bile in these patients suggests that functional derangement of the liver played a role in the production of the symptomatology. The results of observations on the last group mentioned would make it seem wise to consider examination of the duodenal bile before making the diagnosis of a gastrointestinal neurosis.

The favorable effects of intraduodenal treatment, obtained in the most of the patients so treated, were shown by amelioriation of symptoms and changes occurring in the character of the duodenal bile. Such results lead to the impression that the method of therapy is efficacious. Perhaps the most significant findings in support of this impression are the effects on the character of the duodenal bile. The changing of an abnormal to a normal, or nearly normal, type of bile would seem to place this form of treatment on a rational basis. The occurrence of such changes has already led to the conclusion[5] that the therapy actually affects the functional state of the liver; and the results of the present investigation confirm that conclusion.

The fact that the diagnosis of cholecystitis was not confirmed by operative procedures leaves doubt as to the exact number of such patients reported who actually had that condition. But to the clinician the importance of the observations reported lies in the fact that a certain number of patients gave histories ordinarily leading to the diagnosis of cholecystitis and that a goodly number of these patients regained apparently good health under intraduodenal treatment. For these reasons it was considered as rational to allow them to forego immediate operation as it would be in the case of a patient with peptic ulcer who progressed favorably under medical therapy.

From the results of this investigation it seems warranted to draw the following conclusions:

1. There exist a group of patients presenting the symptoms commonly ascribed to cholecystitis, a group with return of symptoms after cholecystotomy or cholecystectomy, and another group complaining of gastrointestinal symptoms not characteristic of any particular clinical entity. The three groups, as described in this communication, are characterized by an abnormal state of liver function, as demonstrated by examination of the duodenal bile.

2. The symptoms presented by the three groups of patients described are frequently relieved during, or following, intraduodenal therapy.

3. Relief from or amelioration of symptoms is accompanied by, or followed by, improvement in the state of liver function, as demonstrated by examination of duodenal bile.

4. Evaluation of disturbed liver function, and its treatment when found, is worthy of consideration in patients presenting the symptoms of cholecystitis; provided the surgical condition of the patient warrants procrastination.

5. Evaluation of the state of liver function is worthy of consideration in ulcer patients who are not progressing satisfactorily, in patients who present unexplained gastrointestinal symptoms, and before making the diagnosis of a gastrointestinal neurosis.

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THE DRAMATIC STORY OF THE NEW HARVARD MEDICAL SCHOOL

BY F. C. SHATTUCK, M.D.

MEMORY harks back to my student days of nearly sixty years ago, to the hideous makeshift of a building which housed the Harvard Medical School, to its squalid surroundings leading Dr. Holmes to apply Campbell's line.—"O Star-eyed Science! hast thou wandered *there*?" Yet therein, thanks to good teachers, to the Massachusetts Hospital next door, and to the City Hospital, young and lusty as an eagle, as good a medical training could be had as anywhere else in the country at that time. Buildings are but shells, necessary; it is the men within who really count. All friends of and workers in the School were overjoyed when in 1883 the new building, corner of Boylston and Exeter, sharing the block with

the Public Library, was completed. It was thought that provision was made for a long future. From the picture of the past I turn to that of the present, and the growth of the School in the last quarter century unfolds itself. But few survive who know the story in full, and I yield to the temptation to put it on record in essentials. I call it a dramatic story. Perhaps romantic would be a better adjective. Both apply, but the former seems better to serve my purpose.

PROLOGUE

An informal meeting at Dr. J. C. Warren's house about twenty-five years ago may serve as the prologue. The facts were patent. First, the Boylston Street building was already outgrown and there was no spare land whereon to build laboratories to meet present needs, far less to meet the inevitable needs of the future. Second, the twenty-five years' nursing of the Peter Bent Brigham Fund was nearing its end, and it seemed possible that some sort of working union between this Hospital and the School might be arranged.

Henry P. Bowditch and J. C. Warren—Moses and Aaron—pondered on these facts, lay awake nights, conspired, and formulated a general plan for the future. This they laid before the meeting, which was attended by President Eliot, I think Doctors Walcott and Cabot on the part of the Corporation of the University, and by ten or a dozen of the professors of the Medical School. The plan involved a large tract of land ample for present and future needs of the school, and also for the Peter Bent Brigham Hospital and other medical undertakings which might be attracted, thus developing a real medical centre.

Bowditch and Warren were just at an age when the visions of the young and dreams of the old should coalesce. They abounded in faith, larger than a cocoanut, and faith carried through. Their dream was as iridescent as it seemed audacious. I, of less faith than even a grain of mustard seed, feared for their sanity. The grandiose ideas of a stage of general paresis were called to my mind. Here was a plan calling for millions. Beyond a limited and restricted endowment the Medical School had not a cent, save what might accrue from the sale of the Boylston Street property. The plan involved coöperation with a Board of Trustees still to be appointed.

ACT I

The first step was to select and secure suitable land. Under the terms of the Brigham will the Hospital must be within the city limits, and the School could not go too far from existing hospitals. Of three possible sites part of the Francis Estate, west of Longwood Avenue, was deemed the most desirable. Not a cent in hand or in sight. Where were several hundred thousand dollars to come from?

Now Henry L. and Francis L. Higginson enter on the scene. To the former belongs the credit of suggesting a sort of syndicate which would buy the twenty odd acres of the Francis Estate and hold the same for five years, during which the School could take it over in whole or in part for cost, interest and taxes. Frank was very active in the successful formation of the syndicate.

> Act I ends with the sight of promised land but no money.

ACT II

An ideal plan of the School, substantially as it now stands, was drawn by Messrs. Shepley, Rutan and Coolidge. Bowditch and Warren, with the help of the late Robert Bacon, his partner, secured an interview with Mr. J. P. Morgan, submitting the plan. "Those are good looking buildings. How much will the administration and two adjoining buildings cost?" he asked. A definite answer not being then possible, he added,—"Send me your architect. Good morning gentlemen!" Several months later, on Class Day, Mr. Morgan cabled from London that he would give the three buildings above mentioned. Later, the other two buildings were ensured by gifts of $250,000 each from Mr. David Sears, and Mrs. C. P. Huntington whose interest was enlisted by her counsel, Mr. Charles H. Tweed, A.B., '65.

> Act II ends with provision toward the desired buildings. The land was finally secured by the gift of their shares by some of the syndicate members, and by money accruing from the sale of the Boylston Street property.

ACT III

Dr. W. B. Coley, H. M. S. 1888, comes on the scene, awakening the interest of Mr. John D. Rockefeller, who sent on Mr. Starr Murphy. He passed a fortnight here making a careful study of the University and of the Medical School in all details. In Mr. Murphy's 30-plus page typed report to Mr. Rockefeller he said,—"Harvard University is a very well managed institution," and advised Mr. Rockefeller to give half a million. Before very long, came a letter from Mr. John D. Rockefeller, Jr., saying that his father was disposed to give a million if three conditions were met:

> 1. He believed that funds on hand and in sight were insufficient to run the proposed school; therefore his gift was conditioned on raising $755,000 by Commencement, about three months off.

2. He would pay his gift proportionately as the sum to be raised was paid.

3. Any portion of the extra sum unpaid within six or eight months—the exact date is not essential—would relieve him of a like proporion of his gift.

His million could be used for land, buildings or endowment, *and no name was attached.*

It has been kept intact for endowment.

Note the different modes of working of the Morgan and Rockefeller minds. Mr. Morgan, in a flash, approves a tentative plan, without interest in detail. Mr. Rockefeller informs himself in full, using the same care in his benevolence as in his business.

May 19, 1904, more than a month ahead of time, Mr. Rockefeller, Jr., was informed by the Treasurer, Mr. Adams,—"We have received $839,361.79 which can be counted towards the sum on the receipt of which your father's gift was conditioned."

Act III thus ends with substantial progress.

ACT IV

Enter the Peter Bent Brigham Hospital. The Corporation was formed in May 1902, with a body of wise and devoted men as Trustees. They bought part of the Medical School land in 1907, built the Hospital, and January 1st, 1913, opened it. They also entered upon a gentlemen's agreement with the University, by virtue of which the *nomination* of Chiefs of Service was to come from the Corporation of the University.

This working agreement led to others similar, with the Massachusetts General, Children's and Lying In Hospitals, for instance. The proximity and example of the Peter Bent Brigham Hospital attracted the Children's and Infants' hospitals, now practically fused, the House of the Good Samaritan, the Carnegie Institute, the Dental School, and, finally, the Lying In Hospital; some on land bought from the Medical School, some on adjacent properties. The development of such a medical centre may have influenced Mr. George R. White and the Trustees of the Angell Memorial Hospital for Animals to build the School for Pharmacy and the Animal Hospital next door. The Psychopathic Hospital nearby must not be forgotten.

Dr. Warren's vision and rarely persuasive powers brought the Huntington (Cancer) Hospital into being, in close union with the Medical School.

Then comes the School of Public Health, made possible by the wise providence of the General Education Board. Into this enter the existing Schools or Departments for the training of Health Officers, for Tropical Medicine, and for Industrial Hygiene. Legally, formally, it is a separate School, with its own Faculty; but practically it may be regarded as part of the Great Medical Undertaking, as of the substance of its mother. Thus provision for the technical training of preachers of the gospel of hygienic righteousness as practitioners, health officers or investigators of the unknown is rounded out.

A wonderful medical centre has come into being and therewith ends Act IV.

ACT V

Dr. Warren again appears at the front of the stage, some part of which he has never left, night or day, for a quarter of a century. Long ago he saw that the medical student was at a serious disadvantage. The School being in Boston the students lacked the provision of dormitories; common meeting grounds outside the class room, laboratory or hospital ward; and provision for the chance of recreation afforded to students in Cambridge by the gymnasium, Soldiers' Field, and Harvard Union. Many of the medical students, all of whom are worked pretty well to the limit, are struggling to get an education with narrow means and under living conditions not promotive of the vigor and endurance their high calling demands. The first public statement of this need was an article by Dr. J. C. Warren in the Boston Medical and Surgical Journal, with a letter signed by the Medical School Class Presidents appended, June 13, 1912. The seed was planted. Signs of germination first showed in the appeal of Dr. Joslin, then President of the Harvard Medical Alumni Association, early in September, 1923, for money for the dormitory. He gathered about him a group of other practical enthusiasts, among whom the untiring secretary, Dr. Francis M. Rackemann, stands out. The Alumni of the Medical School responded generously, as did lay friends. This part of the play is so recent that I can save your patience, simply adding that last spring Mr. Harold S. Vanderbilt, a veritable *deus ex machina*, modestly came on the stage with a promise of a gymnasium and the salary of a director for five years. Unsated in wise liberality, during the summer he promised $575,000 more, thus warranting the completion of plans and the construction of a home for two hundred and fifty medical students, providing lodging, food, suitable meeting and study rooms, a large hall, a gymnasium and tennis courts. True it all is, even if too good to be true. We rejoice not only that the capstone of what Bowditch and Warren saw in their

dreams is about to be placed, but also that Dr. Warren, in this respect more favored than Moses, actually enters into the promised land. We hold Dr. Bowditch, who like Aaron passed on in mid-journey, in grateful and tender memory, and wish that he in the flesh could have shared in this joy.

End of Act V.

But the curtain does not fall. It never will fall on the Harvard Medical School as long as the School lives up to its traditional and present standards.

EPILOGUE

A Medical School unsurpassed in scope and completeness has been brought into being. It must continue to grow, ever a pioneer in keeping ahead of immediate demands. The past carries conviction that the fertilizers, love and money, will be as unfailing as the widow's cruse of oil.

MEDICAL PROGRESS

THE PROGRESS OF NUTRITION

BY FRANCIS LOWELL BURNETT, M.D.

THE INTEGRITY OF THE BODY

THE disorders of the body have been classified according to their known or supposed mode of origin, as infectious diseases, metabolic disorders, and so on. In the infectious diseases an organism can often be isolated from the lesions, so that one etiological agent is specific. With the metabolic disorders however, while there appears to be a disturbance in the digestion of food and its assimilation by the tissues of the body, a conception of the normal anabolic processes has not been proposed. But when the digestive system is thought of as a highly perfected organ which is purposely designed to nourish the body, yet at the same time is very delicately adjusted to internal and external influences like the circulatory and respiratory systems, numerous factors have to be adjusted and controlled to fulfill the normal requirements. From this point of view digestion, absorption, and assimilation are critical processes, which change enormously from time to time without symptoms or generally recognized signs; and on this account the tissues of the body apparently become depleted and metabolic disorders arise. This fact may be readily observed in animals fed on an incomplete food. When the animals first ingest this food a slight and unapparent failure must occur in the anabolic process; but some time elapses before pronounced signs and symptoms of a recognized disorder are apparent.

It is partly because the great intermediate zone between the beginning of ill health and the recognized signs of disease remains unexplored and unknown; but more especially because the signs of health, the factors that promote it, and their practical application, still remain obscure and untried, that the treatment of some of the disorders of the body continues to be very empirical. In an interesting study by Cramer (*Lancet*, I, 633-640, March 29, '24) on the Vitamins and the Borderland between Health and Disease, observations were made on the growth and development of rats on a vitamin poor in contrast with a vitamin rich diet, and while the former animals were generally underdeveloped and weak, they did not show signs of obvious disease, and were able to reproduce. Furthermore, animals on a deficient diet readily succumb to infections. These facts suggest that a reserve of health or an improvised state of well being is acquired by complete and proper nourishment; and factors of safety in the tissues, a strong constitution, natural immunity, etc., are perhaps merely phases of this reserve of health, in which the integrity of the body is slightly—if at all—impaired.

A few persons of today pass through life with few disorders and continue vigorous in their declining years. The ancient Greeks attained a physical perfection unsurpassed by any race. These facts indicate that a reserve of health, and an improved condition of physical fitness is possible. Perhaps the Greeks attained this condition in part by spartan living, inasmuch as they ate only twice a day and exercised their muscles regularly in gymnasia and athletic contests. These measures must serve to promote health. But by a more exact knowledge of how and what to eat, the requirements of the muscles for exercise, the demands of the body for rest, the needs of the mind for

cultivation, etc., a great deal more can be done to keep healthy. For by realizing that there is an unexplored zone between health and recognized disease, that relatively little is known about complete and proper nourishment, but that an improved state of well being is possible, it seems as if the most highly perfected organism must have the inherent capacity for maintaining its integrity.

THE PHYSIOLOGY OF NUTRITION AND THE ANABOLIC PROCESSES

If the development and maintenance of the normal tissues is inherent in the human body, they must be largely brought about by the anabolic processes. But the assimilation of complex substances forming the normal tissues is for the most part dependent on the absorption of digested products from the aliment, and while the complex chemical changes that take place in the cells are generally far too intricate to understand at present, it does seem possible from an understanding of the course of the aliment through the bowel and its final moulding in the form of the feces, to get some knowledge of the anabolic processes and the sustenance of the tissues.

From studies on the form of the feces and the intestinal rate in the physiology of nutrition, the course of the aliment through the bowel, in the complete cycle of digestion and absorption, is a very intricate and sensitive process. In this cycle the alimentary canal does not act merely as a tube, but as an exceedingly complex mechanism. The action of this organ cannot be at fault, but its critical requirements, in the time of eating, the amount consumed, the completeness, proportion and preparation of the food are not often fulfilled. From understanding these requirements, consideration is chiefly given to the digested products that pass through the wall of the canal and into the body, rather than the amount of aliment passing along the canal and through the body. The small intestine, from which most of the absorption is supposed to take place, appears to act generally in a normal way, although the absorption may vary a good deal in accordance with the potency of the digestive fluids. But the colon is so peculiar and so sensitive in its action, that it is not often a useful part of the digestive apparatus; and yet its normal action must be essential for maintaining the integrity of the body.

In this action a pouch or secondary stomach —the proximal colon—forms, with a proper alimentary mixture when the demand of the body is greater than the nutritive supply. From this point of view it seems as if the alimentary canal were intended for two meals, one in the middle of the morning and the other in the evening. But the same action and absorption is generally brought about if the first meal is divided into two small meals, breakfast and luncheon, and a satisfying meal is consumed in the evening. The purpose of the proximal colon appears to be somewhat analagous to the caecum of the herbivora. In these animals, the transformation of coarse vegetable substances into flesh and blood is brought about in part by prolonged digestion in the caecum and proximal colon. As man is an omnivorous animal, some portion of the digestive apparatus must serve in a similar way to transform carbodydrate food into the tissues of the body; and without this action there is a failure of the carbohydrate metabolism. Furthermore, this sensitive mechanism forms the intestinal contents into discrete masses that are readily apparent in the feces.

Investigators of nutrition have not generally given the functions of digestion and absorption the consideration they deserve. By feeding animals on incomplete diets remote effects in the mal-formation of bone, degeneration of nerve tissue, or alterations in the vascular walls have been emphasized. The soft and formless feces occurring early or the diarrhoea in the terminal stages of an incompletely fed animal has not often been recognized. In an illuminating study by Gross (*J. Path. Bact.*, 27: 27-50 Jan. '24) the relation between incomplete diets and the functions of digestion and absorption has been made. He mixed charcoal with the food of rats and observed when it was first and last seen in the feces. With a complete food the charcoal was first noticed in 9 hours, but continued to be seen for 120 hours after ingestion. With a diet deficient in vitamin A the time was 5 and 16 hours; but without vitamin B 16 and 384 hours elapsed. Animals without vitamin A consumed sufficient food but it passed rapidly through the bowels and there was a failure of absorption; whereas animals without vitamin B lost their appetite, ate insufficient food and there was an abnormal retention.

Variations of absorption in animals undoubtedly has an effect on the formation of the tissues. Starved, growing animals without all of the essential food substances do not develop pronounced lesions, because without nutritive material in the alimentary canal absorption is impossible and the anaboiic processes are inactive. But with some kind of aliment for absorption the body apparently makes an attempt to fulfill the requirements of the normal tissues, and failing to obtain the essential substances or through faulty absorption, some kind of material is assimiiated. Abnormal assimilation of this kind has been shown in the teeth of rats on a scorbutic diet by Toverud (*J. Biol. Chem.* 58:583-600, Dec. '23). In these teeth the normal orthodentin is largely substituted by osteodentin and the normal calcium is somewhat replaced by magnesium. The magnesium tooth is much less resistant to the agents that

produce decay, according to Howe, (*J. Am. Dental Asso.*, Dec. '24) for lesions are sometimes apparent which seem to be identical with human dental caries.

Civilized man generally ingests a complete food; consequently the effect of a deficient diet is not often a serious consideration in variations of digestion and absorption. But such pronounced variations occur in man, that other factors must produce a somewhat similar effect. These variations have been shown by Alvarez and Friedlander (*J. A. M. A.*, 83:576-580, Aug. 23, '24). They observed the passage of beads and barium meals through the bowels of apparently healthy young men and women. In general 15% of the beads were dejected the first, 40% the second, 15% the third, and 10% during the fourth day. But in some subjects having a fast rate in which 85% of the beads were dejected during the first 24 hours, the feces were soft and contained considerable undigested food. And then in others that took a week or longer to deject 70% of the beads, no signs or symptoms of ill health were apparent.

In considering the nourishment of the human body from this critical point of view, it would seem as if the treatment of many metabolic disorders—except dietary adjustments—must continue to be very empirical. This is because there is always nutritive material in the alimentary canal, from which some kind of absorption is continually taking place, and some kind of sustenance is being assimilated by the tissues. With slight adjustments in the alimentary mixture advised by the doctor or considered best by the patient, the anabolic processes and the sustenance of the tissues may be greatly improved. If this occurs during the application of some external remedy, the improvement in the disorder may be ascribed to the remedy. Accordingly, it is impossible to ascribe the cure of a metabolic disorder to a simple remedy without recognizing an improvement in the amount and kind of absorption from dietary adjustments.

THE INTESTINAL FUNCTIONS AND RATES

Internal and external stimuli produce enormous variations on the action of the intestine; but the length of the intestines vary a great deal. According to Bryant (*Am. J. M. Sc.*, 167:499-520, Apr. '24) 10% of men and women have a small intestine that varies between 5 and 7 meters; but the extremes for the small were 3 and 8.5 meters, and the large gut 1 meter and 3.25 meters. The myentric reflex by which an intestinal stimulus causes a constriction above and a dilatation below is questioned by Alvarez (*Am. J. Physiol.*, 69:249-253, July '24) from numerous observations on rabbit's intestines. In the same periodical he has also demonstrated "peristaltic rush" with the electro enterogram. The cecocolic tract has been investigated by Hirsch (*M. J. Record*, 119:541-549, June 4, '24) who says "this tract both in lower animals and man, has for its important object the retention of the caecal contents for the necessary complex digestive changes to take place."

The work of Alvarez and Friedlander on the rate of progress of food residues through the bowel has already been alluded to. F. Hoelzel (A. H. Bensch Printing Company, Chicago, 1924) in a pamphlet with the same title discusses the intestinal rate; and then gives the results of the passage of seeds, beads, charcoal, knotted strings, gravel, bone ash, ground nuts, silver wire, through the bowel of one person. He found in general, that the heavier material went through the most rapidly; but as the markers were not taken at a specified time in relation to meals, and meals were frequently omitted during the test, the experimental evidnce is not very conclusive.

Fat digestion, absorption, and assimilation in man and animals by means of the dark field microscope and a fat soluble dye were made by Gage and Fish (*Am. J. Anat.*, 34:1-85, Sept. '24). In this comprehensive and interesting study the authors give a historical account of the subject, measure and enumerate the fat globules (chylomicrons) in the blood, note how the maximum number appear in the peripherical circulation 3-4 hours after the ingestion of the fat, and show that fat is released into the circulation from the body's reserves during a fast. The dye used in these experiments was Soudam III: and while this substance produced a readily observable reaction in the fat of the body and milk of cats, rats, and goats, and also hen's eggs, it was impossible to stain cow's milk.

THE NOURISHMENT OF CHILDREN

The weight—height—age tables as an index of nutrition recently revised by Baldwin and Wood (*Amer. Child Health Asso.*, July 1923) have been given a critical study by Clark (*Pub-Health Rep.*, 39:2199-2208, Aug. 29, '24). He compares the state of nutrition with the height and weight measurements given in the old and new tables, and finds many less children are underweight by the new standard. Baldwin (*Jour. A. M. A.*, 82:1-4, Jan. 5, '24) comments on the use and abuse of these tables as follows: "A deviation of only a few pounds from normal weight is not considered significant, but children under 10 years who are 6% or more under-weight for their height and age, and those over 10 years who are 8% or more underweight for their height and age, are likely to require dietary treatment. Children who are 15% over-weight for their height and age may also be in need of medical attention." A still further critical review of the Baldwin-Wood tables is given by Dublin and Gebhant in a pamphlet (*N. Y. Asso. Imp. Con. Poor*, 1924).

They found when children were not less than 10% under-weight and were then given a thorough examination by a physician for malnutrition that almost half of the children were rejected. The question, however, is not whether the tables are perfect, but if they serve some useful purpose. If they merely serve as an incentive and guide for parents and nurses to keep a check on the growth and development of children, they fulfill their requirements.

A good deal of concern is sometimes experienced with children, who in spite of consuming a large amount of food fail to gain in weight and strength. Growth and development do not depend so much on the amount of nutritive material passing through the body, as they do on the digested products that are absorbed and pass into the body. A child that habitually eats too fast and too much, is frequently upset with indigestion. In this case a factor that plays an important part is the use made of the saliva in digestion. In an interesting observation by Jackson on Saliva in the Nutritive Processes (*Arch. Pediat.*, 40:324-326, May, '23) the value of ptyalin is shown in the nourishment of children with oesophageal atresia. In these patients, an opening is made into the stomach and the child is fed through a tube. At first a sufficient and complete food was poured into the tube, but the children failed to gain in weight or strength. But by getting the patient to spit, or else chew the food first and then spit it into the tube good nutrition was maintained.

OVERFEEDING, MINERAL REQUIREMENTS

The nutritive mixture essential for the proper growth and maintenance of every animal is probably peculiar in some respect. Grafe, Weissmann (*Deutsches Arch. f. Klin. Med.* 144: 350-359, Jan. '24) gave dogs an excess of carbohydrate food and found it produced a decrease in weight but an increase in total metabolism. Although prolonged overfeeding with fat decreased the weight of the animals, it did not increase the metabolic rate. By increasing the basal requirements of patients up and about a ward by 800-1000 calories Kahn and Olmstead (*An. Clin. Med.*, 3:143-148, Aug. '24) found there was an increase in weight of 5-7 pounds a month, but with ten diabetics eight showed an inability to absorb additional food. In these patients, too, the duodenal enzymes were only 25-50% potent, and the feces were filled with indigested food.

The mineral requirements for proper nourishment are receiving more and more attention. Goitre is the most noteworthy disorder of the mineral anabolism. Away from the sea-coast common foods have a relatively low iodine content. Jansen (*Neder. Tijdschr. o. Geneesk.*, 2:348-360, July 19, '24) observes that in Holland 50% more of cases of goitre occur in Utricht than Amsterdam. The inland water may be improved by the addition of one kilogram of iodine to every 100,000 gallons, or if a person takes only 0.05 mgs. of iodine daily, goitre will be prevented. In this country, McLendon and Hathaway (*Jour. A. M. A.*, 82: 1668-1672, May '24) made an analysis of foods from the goitrous regions of Minnesota and Oregon and found the iodine content 100% lower than the foods of New England and California. About the same difference was also found in the drinking water. Foods made from seaweed contain the greatest amount of iodine, but it is also present in fish, milk, butter, leafy vegetables and fruits. In a comprehensive paper on goitre McCarrison (*Brit. M. J.*, 1:989-994, June 7, '24) points out that this disease is prone to occur in fetal life, adolescence, and during pregnancy. Deficiency in iodine is the immediate cause. The deficiency may be due to an insufficient amount in the food, but also on account of a disorder of the bowel, to insufficient absorption.

The number of minerals forming the human body is not generally given a great deal of thought. Orr (*Brit. M. S.*, 2:504-508, Sept. 20, '24) notes that 10 of the 15 elements forming the body are mineral, and each one performs a particular function. Their presence in the food is especially important during development. Green vegetables and egg yolk are rich in iron. Milk, vegetables and some fruits contain calcium. Bertrand and Benzon (*Ann. de l'Inst. Pasteur*, 38:405-419, May '24) fed mice on a zinc free diet and noted that they often lived only half as long as animals on a complete diet. They regard from 0.15-0.30 mgs. of zinc a day essential.

THE VITAMINS

The injurious effects of food-deficiency from the clinical point of view should not, according to Hess (*Atlantic M. J.*, 27:467-469, May '24) be thought of generally as producing specific diseases as scurvy and rickets, but rather as disorders of nutrition which produce slight and manifold disturbances of function. The most pronounced signs of such a deficiency may be readily apparent in the eyes, but with it there may be neuritis, cardiac enlargement and atrophic disorders of the skin, nails, hair and caries of the teeth. The accessory food factors are stored in some of the organs and tissues of the body as a reserve, and therefore an animal while being deficiently fed utilizes the reserve until it is exhausted and then a readily recognized disorder is apparent. The intermediate zone between health and disease has been considered by Cramer (*Lancet*, 1:633-640, Mar. 29, '24). He observed rats on a slightly reduced vitamin diet bore young that were underweight and had diminished resistance to disease. When these animals were put on a vitamin free

diet they rapidly felt the effect, while the young of better nourished mothers continued to increase in weight for a while. Then if the feeding of the former was deficient for two months, it was impossible to get them to improve again.

Various hypothesis have been proposed to explain the action of the accessory food substances. Thus Abderhalden (*Klin. Wochenschr.*, 3:367, Feb. '24) says the vitamins like the hormones exercise their functions by the stimulation of substances in the cells, but the cells must be in the right physical and chemical condition to be receptive. On the other hand, Hess (*Deutsch. Med. Wochenschr.*, 50:163, Feb. '24) observed a reduction of the metabolism in fowls on a diet deficient in vitamin B; and as yeast relieves the signs and symptoms of polyneuritis suggests that the vitamin acts as a catalyser in the mechanism of oxidation. Zilva (*Brit. Med. Jour.*, 1:720, April 19, '24) reduced lemon juice to a concentrated extract, containing all of the vitamin content, which is only 0.5% of the original fluid. In using the extract in a child with scurvy, it was impossible to administer the vitamin content of 15 lemons in the first 24 hours. Although some foods do not contain anti-rachitic substances, they may be made so, according to the work of Hess and Weinstock (*J. A. M. A.*, 83:1845-1846, Dec. 6, '24) by irradiation with the mercury vapor quartz lamp. The observation is of scientific rather than practical value.

CONSTIPATION

Since the publication of "Useful Cathartics" by Fantus (*Amer. Med. Asso.*, Chic., 1920) a few articles have appeared on constipation. Horn (*Texas State J. Med.*, 19:622-626, March '24) discusses the laxative habit as an etiological factor in chronic invalidism. He says in part that the bowel is a very sensitive apparatus, which is greatly influenced by the habits of the individual. The delicate mechanism will stand much abuse, but revolts at insult. When it has been found out that there is no greater insult to the normal operation of the bowel than the laxative habit, there will be less neurasthenia and fewer gastro-intestinal invalids. Burnett (*J. A. M. A.*, 83:996-998, Sept. 27, '24) by an appreciation of the physiology of nutrition as shown by the intestinal rate and the unit form of the feces has shown the injurious effects of catharsis. From this point of view the action of the intestine is not at fault, but the ingested material. Improperly prepared or proportioned food is frequently a breach of the delicate and complex requirements of the body for nourishment, and while an oil or laxative may relieve the symptoms, they are still more injurious by lessening the absorption.

RICKETS

Rickets develops when either the calcium or phosphorus absorption is deficient. Normally the blood serum contains 10 mgs. of calcium and 5 mgs. of phosphorus to 100 cc. Failure of absorption is not often the result of insufficient calcium and phosphorus in the aliment, for Orr, Holt, Wilkins and Boone (*Am. J. Dis. Child.*, 28:574-581, Nov. '24) have shown that with an excess of one of these elements in the diet an insoluble salt is formed which is excreted. Telfer (*Quart. J. Med.*, 17:245-259, April '24) believes that the absorption of calcium is dependent on free hydrochloric acid and is restricted by the alkaline intestinal secretions. In this way the absorption may be limited to the small upper portion of the intestine. The cause of the malabsorption of calcium and phosphorus for the normal development of bone still remains a mooted question. It may be due in some cases to the ingestion of insufficient fat, in others to insufficient sunlight, while in others insufficient exercise may play a part. DeBuys and Von Meysenbug (*Am. J. Dis. Child.*, 27:438-443, May '24) administered one and a half teaspoons of cod liver oil daily to a group of infants and compared the results with a similar study made without giving this fat. Signs of rickets developed in spite of this treatment, although some of the signs were not marked. Mellenby (*Brit. Med. Jour.*, Vol. I: 895-900, May 24, '24) produced typical rickets in puppies on a diet physiologically perfect except for vitamin A, and a diet overbalanced with cereals especially oatmeal prevents calcification in proportion to the amount used. And by the exposure of animals to natural and artificial radiation the amount of calcification was improved.

Findlay (*Jour. A. M. A.*, 83:1473-1479, Nov. 8, '24) discusses the pathenogensis of rickets. The defective demand for calcium may be responsible, on the other hand greater stress may be put on the reaction of the intestinal contents, or a phenomenon which is not yet known in physiology. In a review of rickets in Vienna, Wright (*Canad. M. A. J.*, 14:320-321, Apr. 24) says that craniotabes and the rosary are important and early signs while epiphysical enlargement and delayed closing of the fontanels have not the same value. From the amount of blood calcium and phosphorus the disease may be divided into two types according to which of these elements is especially low.

PELLAGRA

It is interesting to observe in the cases of pellagra reported by Goldberger and Tanner (*Pub. Health Rep.*, 39:87-107, Jan. 18, '24) that most of the patients had diarrhoea. The failure of absorption is presumably due to an excess of carbohydrates, because the addition

of protein in a better proportioned diet relieves the diarrhoea. In the "beef" diet advocated, the protein amounts to about 100 grams, the fat 100, and the carbohydrate 300 grams. An increase in the protein brings about better absorption as the feces become formed (Personal communication), and after a few months of this kind of absorption, the disorder is relieved, and there is a gain in weight of five to eight kilograms. From a study of the diets of people in the cotton mill villages of South Carolina, the authors found that the daily consumption of 30 gms. of protein or 500 cc. of milk was usually sufficient to prevent pellagra. The importance of milk in the diet of the Southern poor is strikingly illustrated by Wheeler (*Pub. Health Rep.*, 39:2197-2199, Aug. 29, '24) in the report of a few families. These people were generally free from pellagra as long as they had a cow and an abundant supply of milk, but when the cow went dry or was lost, pellagra appeared in many members of the families.

SCURVY

A very comprehensive and abundantly illustrated report was made by Hojer, (*Acta Paediat., Supp.*, 3:8-278, '24) at the Karolinska Institution in Sweden. The author gives a short historical review of the subject, and then a very detailed account of the lesions produced in about a hundred guinea pigs. A few of the animals were fed on a basal diet of oats, bran, milk and ol. jecoris Asuli; while all of the others had supplementary diets of turnips (controls), whortleberry, strawberry, orange, raspberry and tomato juice in varying amounts and conditions. On this basal diet the tomato and orange juice were the most effective in healing the lesions of scurvy. In the study of the pathology of the disorder, he says that changes are brought about in most of the tissues. In the development of bone, there is a degeneration of the bone forming cells, as well as an atrophy of the connective tissue which produces a symptomatic effect in the vascular system. Atrophy, and necrosis too, were seen in the lymph tissue, muscles, and glandular organs. In a supplementary section the relation of scurvy and tuberculosis is studied. The infectious disease may inhibit the ingestion of anti-scorbutic food by a poor appetite, or change the absorbing power of the intestine. But the failure of normal and strong connective tissue to wall off the lesions makes the infectious disease more rapidly fatal.

In determining the effect of acute scurvy on the subsequent nutrition and growth of guinea pigs, Anderson and Smith (*J. Biol. Chem.*, 61: 181-191, Aug. '24) fed young animals on a soy bean, cod liver oil, yeast and salt mixture and produced the disease. The animals lost their appetite on the scorbutic diet, and there was a decrease in weight; but when raw cabbage was added, more food was consumed and an accelerated rate of growth ensued. On the other hand, Gerstenbeger, Champion and Smith (*Am. J. Dis. Child.*, 28:173-182, Aug. '24) tried the effect of pregnancy on the course of scurvy in guinea pigs. The females were given oats, milk and orange juice, and put with the males. At the end of four weeks half of the females were found to be pregnant, and the orange juice was left out of the diet of all the animals. About 70 per cent of the young were dead at birth and showed varying degrees of scurvy. The adult animals only lived a few weeks. On examination the pregnant animals showed relatively little evidence of scurvy, but in the non-pregnant females the signs of the deficiency disorder were marked.

A case of human scurvy is reported by Cain (*Rev. De Med.*, 41:36-46, '24) in which the preliminary symptoms were dyspepsia and emaciation. By the addition of milk, vegetables and fresh fruit, the scurvy was relieved, but the other symptoms continued. Schagen (*Jahrb. f. Kinderh.*, 104:225-238, Feb. '24) studied scurvy in children during the famine of 1918-1919 in Russia. The incidence of the disease in 1913 was only .073 per cent of the inhabitants of St. Petersburg; whereas in 1920-1923, 5.2 to 11.3 per cent of the patients examined at the children's clinic had scurvy. The food of these children was sufficient, but was almost always without fruit and vegetables. The disease was found to involve many organs. Stomatitis was gangrenous in 13 per cent of the cases, severe anemia generally present, and many patients had latent tuberculosis as shown by the Von Pirquet test.

BERIBERI

The relation of rice to beriberi continues to receive a good deal of study. Cobb (*Indian M. Gaz.*, 59:401-402, Aug. '24) observed that when there was a rice shortage in Malaya that beriberi almost disappeared, because wheat, flour and green vegetables were consumed in large quantities to replace the rice. But when rice became abundant again, beriberi became very evident. Shinoda (*Zeitsch. f. Klin. Med.*, 100:151-169, May '24) gave two healthy persons a diet without Vitamin B for four months, but beriberi did not develop. Oedema, tachicardia and hyperasthenia were evident. From this fact the author is inclined to believe that beriberi is due to an excessive carbohydrate diet. McCarrison (*Brit. M. J.*, 1:414-420, Mar. 8, '24) says that people living on decorticated rice for any length of time develop beriberi; but if the grains contain 0.4 per cent of phosphorus pentoxide the disease will be prevented. He also (*Brit. M. J.*, 1:567-569, Mar. 29, '24) tried feeding pigeons on a diet without Vitamin B to which millet grown with cow manure, chemical fertilizer, and on poor soil,

was added. Manured millet prevented neuritis the longest.

An interesting account of an outbreak of beriberi in Freetown, Sierra Leone, is given by Blacklock (*Brit. M. J.*, 1:1046-1047, June 14, '24) in which lack of exercise played an important part. Of the 59 patients, 34 were tailors who habitually led a sedentary life. To prove the hypothesis he fed two groups of fowl on polished rice. One was closely confined, and the other allowed to exercise. The latter group lived almost twice as long as the first.

VITAL RESISTANCE AND TISSUE REPAIR

A loss of vital resistance in animals on deficient diets has been generally observed for sometime, but Werkman and others (*J. Infect. Dis.*, 34:447-453, May '24) made a special study of the action of doses of B. Typhosis and B. Anthracis in guinea pigs with acute scurvy. The susceptibility of these animals to infection was greater than the normal controls, and follows in general the lowering of the body temperature. Somewhat the same results have been found by Webster and Pritchet (*J. Exper. Med.*, 40:397-404, Sept. '24) in mice. They tested two groups of animals with mouse typhoid bacilli, mercury bichloride, and botulinus toxin. One group had been fed on bread soaked in milk, with supplemental feedings of oatmeal, buckwheat mixture, and dog biscuit; the other group was fed a diet of 67.5 per cent wheat, 15 per cent casein, 10 per cent milk powder, 5 per cent butter fat, sodium chloride 1 per cent, and calcium carbonate 1.5 per cent. While the growth and development of both groups appeared to be quite similar, the animals in the latter group were more resistant to infection and poisoning than those in the former.

The repair of tissue appears to be influenced by the nourishment of the animal. Moise and Smith (*J. Exper. Med.*, 40:13-23, July '24) fed albino rats on a high protein, high fat, high carbohydrate, and well balanced diets; and destroyed portions of the liver by injecting similar amounts of chloroform. The animals were killed at the same time after the injections and the lesions studied. The tissues of the animals on a well balanced and high protein diets showed considerable resistance to the injurious agent; and the least amount of repair occurred in rats on the high fat diet whereas the reparative process of the animals on the high protein and high carbohydrate was about the same. The well balanced diet brought about the most rapid and complete regeneration.

A study of bone forming elements in the blood in ununited fractures was made by Petersen (*Bull. Johns Hopkins Hosp.*, 35:378-381, Nov. '24). He first determined the calcium and phosphorus content of dogs' serum on a normal diet, and then gave the animals three diets, one normal, one low in both calcium and phosphorus, the other low in phosphorus and high in calcium. On the deficient diets the phosphorus was reduced about 50 per cent. The bones were broken and callus formation and calcification observed with the roentgen ray. The bones of the dogs on a deficient diet remained ununited after four weeks, whereas the bones of animals on a complete diet with normal phosphorus were well united at the end of this time.

THE PRINCIPLES AND PRACTICE OF HEALTH

In the previous sections experimental evidence has shown that complete nutritive materials must be ingested if the normal nourishment and health of an animal is to be maintained. While experimental animals require a comparatively simple food, comminute it when necessary, and do not over-eat, man lives so much by custom and habit that there are many more factors to take into account in adjusting the diet to the nutritive requirements. But when an adjustment is made by which the food is complete, well proportioned, without an excess, and thoroughly masticated, the intestinal contents will be moulded into discrete masses or fecal units which are commonly dejected twice during a day, and the passage of a meal through the bowel takes about 62 hours to appear and 134 hours to disappear from the feces.

Assuming that this form of the human feces and the intestinal rate are indices of normal absorption, health and disease may be considered from a new and a somewhat more enlightened point of view. And instead of being ignorant or careless in what and how to eat, in how to exercise and rest, etc., we must generally strive to control and regulate these factors in order to keep the feces and intestinal rate normal; for under these conditions only may the anabolic processes become complete, and the integrity of the body generally maintained. In considering this point of view from our knowledge of the accessory food factors alone, there are not many who would intentionally and habitually consume an incomplete food, and become afflicted with rickets, beriberi, scurvy, etc. This is because the necessity for a complete food, that is one containing all of the vitamins, has been generally recognized. But when the diet of man is considered from a more critical point of view, there are faulty food factors that influence the physiology of nutrition in a somewhat similar way. These faulty food factors, such as eating too fast and too much, too much rich and complex food, and the ingestion of an oil or drug that prevents normal absorption, have very properly been called Mortamins (*Burnett*, BOSTON MEDICAL AND SURG. JOUR., 188:987-904, June 7, '23).

In realizing that the avoidance of these factors is conducive to an improved state of well

being, it is necessary to educate the patient in the practice of health. Since the form of the feces evidently affords a means of determining normal digestion and absorption, it is first necessary to tell the patient how and what to eat in order to elicit the normal action of the gut. For this purpose the patient should keep a record of the food, the time spent at meals, the time and kind of dejection and a determination of the intestinal rate. Additional information of the amount of exercise or rest may also form part of the record. The first record generally shows a rapid intestinal rate; and this may be due to improper preparation, too much food, an excess of carbohydrate, too much rich food or fruit. These dietary indiscretions should be corrected during the following week and then a full record kept for the third week. This may show a slight improvement in the absorption. At this time the patient may have learned that too much mayonnaise dressing, or grape fruit and cranberries, too many tomatoes ingested one day, decrease the absorption. An excess of fat or sweet foods may similarly increase the rapidity of the rate and cause intestinal indigestion.

When the patient has learned the factors in the diet that prevent intestinal indigestion and generally controls them, finds out the therapeutic value of muscular exercise and takes it, a determination of the intestinal rate should be taken once every month and kept at about 62-134 hours. At this time too, the body should be accurately weighed and recorded. If these determinations are about right, and there appears to be a general improvement in the conditions of the body, further advice is unnecessary for a few months. On the other hand, if intestinal indigestion occurs every few days, or the presumably normal intestinal rate cannot be maintained, a full record should be kept every two or three weeks and further advice sought from the counsellor of health.

When the treatment has brought about a general improvement in the body, a comprehensive and detailed health examination should be made. At this time the patient may feel very much improved, be without symptoms or signs of a previous disorder; yet in a careful and thorough health examination abnormal conditions will be revealed. He may still be obese, have a blood pressure of 65/100, and a haemoglobin of 84 per cent (Haldane Scale) or a blood sugar of 0.140 per cent. Furthermore, so many of the metabolic disorders are likely to recur, that the patient should be given exact information about his condition, and urged to continue his efforts to keep well in order to prevent another attack, for it seems quite possible that the only cure of some of these disorders is the maintenance of health.

If the human body has the inherent capacity to maintain its integrity, the result must be largely brought about through the anabolic processes and the sustenance of the tissues. The anabolic processes, however, are influenced by many factors; and while the amount and kind of absorption derived from the food is undoubtedly the most important factor, in early and slight changes in these processes, the indices of absorption are not often observed. Indeed, relatively little is yet known about the form, reaction, and composition of the normal feces; and a great deal still remains to be learned about the normal intestinal rate. Our knowledge is still meagre in regard to the value of muscular exercise on the general fitness of the body. Rest is an important factor in the relief of some of the diseases, but there is more to learn about its relation to the maintenance of health. Some advances have been made in understanding the requirements of a healthy mind, and so on. Indeed, the equilibrium of the body in health is in a very complex and delicately adjusted state. But how is it to be maintained in this condition? With the increasing demand of the public for instruction in health measures, more must be learned and more must be taught. In the near future it seems not unlikely that Health Institutes will be established, where the people will be educated in the use and care of their own bodies, in order to live; and at the same time through special studies and investigations a great deal more will be learned about the principles and practice of health.

WEEKLY NOTES ON CHILD WELFARE TOPICS COMPILED BY THE U. S. CHILDREN'S BUREAU

PURE MILK, ILLINOIS

CERTIFICATES of approval from the Illinois Department of Public Health must be secured by all milk pasteurization plants, except in large cities with good municipal inspection, according to a recent law. Employees in such plants must have annual physical examinations.

BABIES SAVED, BRIDGEPORT, CONN.

Four years' work by Bridgeport's division of infant hygiene has reduced the city's infant mortality rate from 92 per 1,000 live births to 56, a drop of almost 40 per cent.

Health stations have aided in this work. The cost of keeping in touch with a baby at home and in the health station is only 29 cents a visit.

WEST VIRGINIA IN THE BIRTH-REGISTRATION AREA

West Virginia is in the 1925 birth and death-registration area of the United States, the Census Bureau announces. The birth-registration area now includes 33 States and the death-registration area 41.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 11491

MEDICAL DEPARTMENT

AN unmarried American woman thirty-eight years old entered September 3 by the recommendation of a physician who asked for study of anemia. Her chief complaint was weakness. She gave a history of yellow fever when she was in Mississippi during an epidemic at the age of ten. She first noticed shortness of breath the winter before admission while she was swimming in Florida. Her catamenia were usually irregular.

In June, three months before admission, she first noticed that she tired easily. Soon afterwards the glands in her neck began to swell. A month before admission she had rather profuse bleeding from the sockets of two teeth after extraction, and had a great many black and blue spots under the skin. No other member of her family had shown this symptom. In August she began to have pain in the back, aching in the right arm and numbness of the forearm. These symptoms improved in a week or two. August 9 her physician found her with a temperature of 102°, rapid pulse and anemia. The fever had continued. A blood culture was sterile. Since July she had had loss of appetite. She was getting weaker every day. She thought she had lost considerable weight. Since the onset of the illness she had noticed dyspnea on exertion. For three weeks she had had increasing sweating. At her last menstrual period there was excessive bleeding.

Examination showed a fairly well developed, slightly emaciated woman with marked asthenia, atonic pale skin and pale mucous membranes. The sclerae were bluish white. In the left anterior and posterior angles of the neck was a mass of large firm discrete lymph nodes. The tonsils were hypertrophied. There was enlargement of the supraclavicular nodes on both sides and marked enlargement of the left axillary nodes. Those in the right axilla were just palpable. The pharynx showed small purpuric spots or petechiae. There were remnants of possible purpuric spots on the legs. There was some oozing from the gums. The apex impulse of the heart was felt in the fifth interspace 8 cm. from midsternum. The location of the midclavicular line is not recorded. The left border of dullness was 9½ cm., the right border 3 cm., the supracardiac dullness 6 cm. The action was regular and rapid. There was dullness at the apices of both lungs and slight dullness at the right base. There were occasional inspiratory râles, not persistent, at the right apex. The liver dullness extended from the fourth interspace to 3 cm. below the costal margin, where the edge was felt. The spleen and both kidneys or masses resembling them were also palpable. There was resistance in the epigastrium. The knee-jerks were slightly hyperactive. The fundi showed several retinal hemorrhages and two gray patches of exudate. The disc margins were blurred. No elevation was made out.

The temperature was 97.8° to 102.5°, the pulse 96 to 147, the respiration 22 to 36. The urine was pink at one of seven examinations, cloudy at two, amount normal, specific gravity 1.010 to 1.022, the slightest possible trace to a very slight trace of albumin at two examinations, 4 leucocytes per high power field to an occasional leucocyte at three, 2 red cells per high power field to a sediment loaded with red cells at three. The blood picture was subject to considerable discussion. The leucocyte count was 72,900 at admission, falling to 4,500 following X-ray treatment and increasing later to 12,500. The smear showed a large number of cells said by some to be of the myelocytic series, and by others to be of the lymphocytic series; by still others they were unclassified. The smear was looked at by Dr. Thomas Buckman, who thought that the majority of the cells were of the lymphocytic series. The hemoglobin was 30 to 45 per cent., the red count 1,380,000 at entrance, maximum 2,250,000 after transfusion, falling to 920,000 the day before death; slight to moderate anisocytosis, poikilocytosis and achromia. The platelets were reduced, twice nearly absent. A Wassermann was negative. The clotting time was 3-10 minutes. Clot retraction was normal. Serum dilution was 1/32.

The patient was given X-ray treatment September 5 in two sittings. Following transfusion of 600 c.c. that day the white count dropped from 25,200 to 19,500, she became less restless and dyspneic and thought she was improving. There was slight bleeding from the gums. The pain in the lumbar region radiating to the legs recurred, requiring morphia. After this she had vomiting which she ascribed to the morphia. She was therefore given luminol instead. September 8 another X-ray treatment was given. There was no reaction. September 14 another transfusion of 600 c.c. was done. Very slight bleeding from the gums persisted. September 17 she was given a quarter erythema dose of high voltage treatment over the left neck without result. The total X-ray treatment was slightly less than half an erythema dose of copper filtrate radiation. Following the treatment the enlargement

of the glands decreased. The spleen was unchanged in size. September 22 she died.

Note by the senior house officer, Dr. D. R. Higbee. The presence of the very large percentage of very young cells in the blood was differently interpreted by many observers. Three attempts to show the presence or absence of oxydase granules were unsuccessful.

Discussion

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

Pernicious anemia goes with fever often. The absence of any obvious cause for anemia here makes us think of that. She is rather young for it, and we usually do not have purpura with pernicious anemia. Moreover the swelling of the glands in the neck is not in any way characteristic of it, and nothing is said of symptoms on the part of the tongue, fingers and toes, which are usually present in pernicious anemia.

NOTES ON THE PHYSICAL EXAMINATION

We do not know the diameter of her chest. The caliper method of measuring the chest has not as yet come into general use in this hospital.

The temperature is elevated most of the time, averaging 100°. It does not touch normal but once in the first fourteen days, and it is still higher on the second sheet, where it averages 101.5°. I do not seem to remember a case of pernicious anemia dying with fever. I have seen a great many have fever, but the cases that I remember came down at the end. I have an idea that it gets down.

Dr. Means: We do not see many cases of pernicious anemia die in the hospital. They are apt to go home.

Dr. Cabot: There was evidence of hemorrhage from the kidney three times; the rest of the time there was nothing in the urinary examination.

There is a tremendous amount of talk about these mononuclear blood cells nowadays, it seems to me, with very little progress over what we knew twenty or thirty years ago. I know perfectly well that I should have called these lymphocytes.

I will bet that the oxydase reaction was absent.

DIFFERENTIAL DIAGNOSIS

I see no reason to make any diagnosis other than lymphoid leukemia, or lymphoblastoma.

When leukemic patients begin to bleed under the skin they always die. I think if that had been realized they probably would not have X-rayed her. I have never known any exception to this statement. They may go along fairly well despite many symptoms and with just as bad a blood as this, but when they begin to bleed under the skin that is the end. Whether that means in all cases an infection or a spread of the disease I do not know. Certainly in many cases which I have seen necropsied there has been a terminal infection which we supposed connected.

Dr. Means: Isn't it connected with the low platelets? They may bleed when their plates get down, when the leukemic process has so encroached on their marrow that they cannot produce platelets any more. I think leukemic patients very frequently have purpura when they are not having infection of any kind. I think it is a blood platelet affair. The anemia is interesting too. I suppose the whole thing is what is called a myelophthisic anemia. The bone-marrow is crowded out by leukemic infiltration, cannot produce plates, cannot produce red cells, does not produce normal white cells, but these abnormal ones.

There is another interesting point, the temperature. It is worth mentioning in passing that in both kinds of leukemia there is an elevation of metabolic rate and the leukemias together with hyperthyroidism are the two afebrile disturbances always accompanied by increased metabolism. Dr. George Minot looked up a series of cases and found that if one really searched one found that leukemic patients have many of the same symptoms that hyperthyroid people do. They want fewer bedclothes and sweat more, because they radiate so much heat.

Dr. Cabot: They both have itching. I do not know whether that is connected with that.

We have no reason to go wrong in the diagnosis here. We have had a biopsy through the blood, and we have no reason to be in doubt. Necropsy ought to show enlarged lymph glands in the usual sites, that is either bronchial or retroperitoneal or both. The spleen and liver ought to be enlarged, and the bone marrow ought to show characteristic changes. Whether there is any terminal infection in this case or not I do not know. We do not need to suppose it. Leukemia, like pernicious anemia, often produces high temperature. But infection is common statistically in cases of this kind.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Acute leukemia.
Lymphatic secondary anemia?

DR. RICHARD C. CABOT'S DIAGNOSIS

Lymphoid leukemia.
Terminal infection?

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

   Lymphatic leukemia.

2. *Secondary or terminal lesions*

   Lymphoma of the kidneys, the liver and the retroperitoneal glands.

Leukemic infiltration of the spleen.
Hemorrhagic edema of the lungs.
Hemorrhages of the epicardium.
Hemorrhages of the uterus.

3. *Historical landmarks*

Polypi of the uterus.
Partial duplication of the left ureter.

DR. RICHARDSON: This case was one of lymphatic leukemia. There were lymphoma of the kidneys, liver and retroperitoneal glands, leukemic infiltration of the spleen, hemorrhages of the epicardium, and some slight hemorrhage in the uterus. There was hemorrhagic edema of the lungs, and there were hemorrhages in the pleura. No terminal infection of any sort was recorded.

DR. MEANS: Did you get any bone marrow?

DR. RICHARDSON: Apparently they did not. I did not do this case.

CASE 11492

MEDICAL DEPARTMENT

A laborer forty-nine years old entered September 9, semi-stuporous, for study of "stomach trouble" of two months' duration. He was slightly irrational and spoke slowly and with difficulty. The history is therefore incomplete and none too reliable. One sister died after an operation for some brain trouble. In boyhood the patient had a discharge from the left ear. At thirty-four he had "asthma."

July 4 he began to develop constant pain all over the stomach, worse after sunset and after eating heavy food. It was relieved by medicine given by a physician. For six or eight weeks he had urinated four or five times at night and had had slight precordial pain. There was some tenderness in the right upper quadrant. For six weeks he had had anorexia, light colored stools and a heavy, stupid feeling in his head. For five weeks he had had increasing difficulty in breathing. For a month there had been slight pain on micturition. For three weeks he had had a yellow tint to the skin and orthopnea. He had had night sweats and chills. Since the onset of the illness he had not slept well. He thought he had lost twenty-five pounds in six weeks.

Examination showed a fairly well developed and nourished man, weak, pale and apparently cachectic, with an atonic facial expression and slurring speech. The mucous membranes were pale. The sclerae were subicteric. The upper teeth were false, the lower teeth in bad condition. The tongue was very markedly atrophied. The mouth was very dry. There were râles at the bases of both lungs, especially on the right. The apex impulse of the heart was not found. The left border of dullness was 9.5 cm. from midsternum, 1.5 cm. outside the midclavicular line. The right border of dullness was not determined. The supracardiac dullness was 5.5 cm. The action was regular. The sounds were of poor quality. The blood pressure was 95/65 on admission, 120/60 later and 100/? still later. Electrocardiogram showed normal rhythm, rate 55, slightly prolonged auriculo-ventricular conduction (P-R interval 0.21-0.22 second), T_2 low, Q-R-S complexes not remarkable. The skin over the abdomen showed a few small punched-out scars. The abdomen was distended, with shifting dullness in the flanks and some generalized tenderness, especially over the liver. The liver was enlarged to 9 cm. below the costal margin. The edge was not felt. The bladder was enlarged half way to the umbilicus. The dorsal gland at the root of the penis was palpable. Rectal examination was negative. The pupils were contracted, otherwise normal. The left ankle jerk was not obtained. There was slight edema of the feet.

The temperature was 97.7° to 102.5° by rectum, the pulse 74 to 105, the respiration 23 to 33. The urine showed a very slight trace to the slightest possible trace of albumin at all of three examinations, specific gravity 1.010 to 1.022, amount normal when recorded, 1-6 leucocytes per high power field at all examinations. At a renal function test the appearance time was two hours; off color. There was a residual of from 350 c.c. to 60 c.c. The blood showed 36,000 to 28,300 leucocytes, 90 to 84 per cent. polynuclears, hemoglobin 50 per cent., 2,590,000 to 2,690,000 reds. Two smears showed slight achromia. A Wassermann was negative. September 9 the non-protein nitrogen was 63 mgm., September 11 the fasting non-protein nitrogen 58 mgm., the CO_2 combining power 29.7 volumes per cent. A blood culture was sterile. A stool examination was negative for occult blood.

The patient was not in continuous pain. His face had a pinched, septic appearance. An enema and turpentine stupes had little affect on the distension or the abdominal resistance. One surgical consultant did not believe that the cause of the symptoms was localized intra-abdominal sepsis of the usual type and advised expectant treatment for the present. Another believed there was an infection somewhere in the abdomen, and though he was doubtful if anything could be accomplished by operation he could see no great harm in an exploratory incision in the right upper quadrant under novocain anesthesia.

By September 12 the patient had little or no pain, but was gradually losing ground. The distension increased. The liver dullness is said to have decreased. September 14 he died.

Note by the senior house officer, Dr. D. R. Higbee. The patient gave a history of abdominal pain of gradual onset present almost daily

for two months. In the hospital there was no real abdominal spasm, but from entrance until death there was moderate resistance and tenderness over the entire abdomen, maximum over the right upper quadrant. On the last day there was great distension not affected by enemas or turpentine stupes.

Discussion

BY DR. MAURICE FREMONT-SMITH

NOTES ON THE HISTORY

Of course this is just the sort of case where an accurate history is of the greatest importance, for here we have already the brain suggested as the seat of the trouble, and a history of an old ear trouble. We should want to know a thousand questions about the immediate past.

There are a good many possibilities here but certainly no story that one can draw conclusions from. Jaundice, chills, and pain stand out in my mind as indicating possible liver infection. The frequency of urination and pain suggest the prostate or the bladder. His difficulty in breathing of course suggests a cardiovascular situation. I do not see how we can bind up any of those possibilities with his stuporous condition unless it be a uremia, and if it be a uremia of course we have to have some other explanation for most of the symptoms in that paragraph.

NOTES ON THE PHYSICAL EXAMINATION

The heart is perhaps somewhat enlarged.

We do not know how thick a chest wall he has, so we do not know how to interpret the heart sounds. If he were thin and cachectic, that is an added factor in our diagnosis of heart failure. Especially is that true if the first sound is of poor quality and the second louder at the apex. Dr. Levine some time ago pointed out that in coronary thrombosis the first sound is often poor, the second sound being good. That taken in connection with other signs,—severe pain, perhaps pericardial friction rub, slight temperature, and an evidently damaged heart—making a picture very suggestive of coronary thrombosis. Coronary thrombosis can develop without pain.

The upper limit of normal for the A-V conduction time is .20. This is very slightly lengthened, and T_2 is low. Those two things together would make one think that the patient had probably been given digitalis. They would both be explained by moderate digitalization. With marked digitalization the T wave is inverted.

There is no interventricular block and no evidence of myocarditis so far as the electrocardiogram can show it.

They do not specifically say that the prostate was negative. Of course we have to think definitely about the prostate. Why do we have obstruction, pain, and frequency?

Miss Painter: The prostate was negative.

Dr. Fremont-Smith: Of course there may be a median lobe which might not be felt by rectal examination and yet which might obstruct outflow. The man may have an old stricture.

So we have a big liver and the question of a cardiac situation. Cirrhosis of the liver of course would explain the ascites, would explain the large liver and the mental condition. Because in cirrhosis of the liver one may have delirium, coma, even changes in the central nervous system giving a Babinski,—in other words, very marked evidence of central nervous system involvement. It would not however explain chills, fever and dyspnea. On the other hand if we look at it from the other end and think of the heart, there can hardly be enough in the heart with an electrocardiogram of this type, with a pulse when he first came in of seventy-four and regular, to account for a liver as large as this and ascites. We would normally have symptoms to go by, but we have very little here symptomatically. I feel that the heart can pretty well be thrown out as the cause of the most important symptoms.

He has a very definite temperature starting at 99° and running up the first night, more temperature than we get with cirrhosis of the liver. We may have a little, 100° to 100.5°, but I have never seen 102° with cirrhosis.

It is very unlikely that we have a chronic nephritis here. We know that he has a large bladder residual, so we are not getting a real renal test here. On the other hand marked retention and back pressure can cause the function to fall to zero, and can, if of sufficiently long duration, cause uremia.

Evidently this man has a pyogenic infection. Could this white count be due to a secondary anemia? It is too high. Twelve to fourteen thousand could be, but not so high as this. The anemia is more apt to be secondary to his infection.

The non-protein nitrogen is very easily explained by back pressure on the kidneys. That is a frequent situation that comes up in dealing with prostates, met by gradual drainage of the bladder over long periods, often bringing the non-protein nitrogen down to normal, making the case suitable for operation.

Why was the diaphragm high on the left? Of course with a subdiaphragmatic abscess we should have an immovable or very slightly movable diaphragm above the abscess. They were evidently thinking along the same lines, that there must be pus somewhere, and were looking for it beneath the diaphragm.

DIFFERENTIAL DIAGNOSIS

I believe this man had intra-abdominal sepsis, liver abscess or multiple abscesses of the

liver secondary to infection elsewhere in the abdomen, perhaps an appendix, perhaps a gall-bladder. Of course liver abscess in these parts is an unusual diagnosis. In the tropics it is very frequent, secondary to amebic dysentery.

He has a big heart, and we shall probably find some of the signs explained by the heart. If one of the abscesses of the liver, if there be such, pressed upon the common duct and caused jaundice it might well have pressed upon the portal vein and caused ascites. It would seem to me very unusual to have ascites secondary to cardiac disease due to a bad heart without more evidence of edema elsewhere, and edema at the bases of the lungs.

I believe that liver abscess is the best diagnosis, and he probably had besides prostatic hypertrophy or stricture. He had also a big heart and ascites.

A PHYSICIAN: How would abscess explain his mental condition?

DR. FREMONT-SMITH: Sepsis.

A PHYSICIAN: I have recently seen a case of perinephric abscess that puzzled me.

DR. FREMONT-SMITH: With central nervous symptoms, with the sort of thing this man had?

A PHYSICIAN: Not exactly.

DR. FREMONT-SMITH: Of course we have not enough to make a diagnosis of his brain lesion, whatever it be.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

General peritonitis.
Liver abscess.
Empyema, gall-bladder.
Malignancy with secondary infection.

DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Abscess of the liver, secondary to appendix or gall-bladder infection.
Ascites.
Hypertrophy of the heart.
Prostatic hypertrophy.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

Gangrenous appendix with abscess, walled about by peritoneal adhesions.
Pylephlebitis with abscesses of the liver and localized perifonitis.

2. *Secondary or terminal lesions*

Soft hyperplastic spleen.
Icterus.
Acute pleuritis.
Edema of the lungs.
Slight hydropericardium.
Ascites.

3. *Historical landmarks*

Chronic pleuritis, left.

DR. JOHN S. LAWRENCE: There was slight icterus. The peritoneal cavity contained about 1500 c.c. of thin clear yellowish fluid,—in places localized peritonitis around the abscesses which were found in the liver in the region of the right lobe. The appendix was lost within a mass of adhesions. Portions of the thickened wall were made out. The lower end of the ileum was coiled up with adhesions continuous with the adhesions about the appendix. The esophagus and stomach were negative. The small intestine showed nothing unusual. The mucosa of the large intestine showed some reddening in the region of the upper half of the rectum, some of the vessels containing some fluid purulent material.

The liver was considerably enlarged,—3190 grams. (Normally 1200-2400.) In the right lobe were two large abscesses which were walled off, showing localized peritonitis in the region over them. But there was not a generalized peritonitis.

The gall-bladder and bile-ducts were negative. The spleen weighed 370 grams, moderately enlarged. The tissue presented as a soft, purplish brown-red mush.

The portal vein and great radicles laid open presented as great drains filled with semi-fluid purulent material.

There was a few c.c. of thin purulent material in each pleural cavity. There were no pleural adhesions on the right. On the left, general pleural adhesions except in the region of the apex, which was free. The lung tissue was spongy, pale to dark reddish, and yielded thin reddish frothy fluid. The pleura over the lower lobe of each lung was coated with exudate.

The heart weighed 377 grams, full sized, the myocardium of good consistence, brown red. Nothing abnormal was found in the heart. The aorta and great branches were negative.

The kidneys were negative.

Culture of blood from the heart showed a few diplococci and bacilli.

DR. FREMONT-SMITH: Did you see the patient in the wards? What was thought of his mental condition?

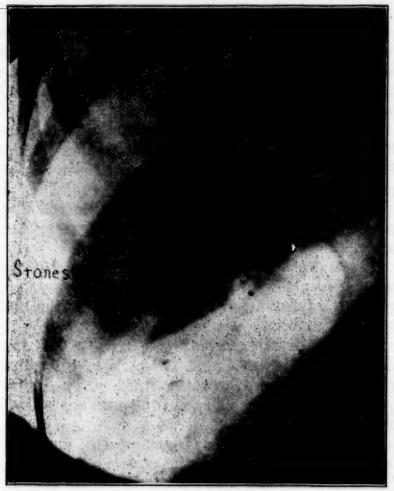
DR. LAWRENCE: He was just an acutely sick patient who could not concentrate long enough to give a good story. We tried to get a story that would be consistent with an old appendix, but from him it was not possible. I don't think he was upset mentally otherwise than that he was too sick to tell us much.

CASE 11493

SURGICAL DEPARTMENT

A married Nova Scotian woman forty-five years old entered April 29 complaining of hemorrhoids and pain in the back. Her mother died of "tumor of the uterus," her father of

cancer of the stomach. She had been married twenty-three years, but had never been pregnant. At twenty-five she had a short attack of colitis with some bleeding by rectum with much pain, and two years later a similar attack. At thirty she was treated for arthritis in the Out-Patient Department of this hospital. She had rare sore throats and rare headaches. She had at times been anemic because of loss of blood from hemorrhoids. At such times her skin became somewhat yellowish and her gums and eyes white, and "if she pricked her finger she would get just water." She had polyuria when nervous or excited. At thirty-nine she had an operation at a Brookline hospital with



Shows shadows which were pronounced undoubtedly gall-stones.

removal of the appendix and she did not know what else. Her bowels had always been constipated. Two years before admission she weighed 193 pounds. After dieting she weighed 178 pounds the January before admission.

For fifteen or twenty years she had had hemorrhoids. For years she had passed considerable bright red blood at stool about once in three or four weeks. Owing to her constipation her bowels moved with great straining and sometimes not for two or three days. For the past two or three years she had taken a great deal of American oil. Beginning a year before admission she had attacks of pain in the back, chest, below the angles of the scapulae, radiating around both sides, brought on by the bending position. (She did much sewing.) The attacks occurred at first once in seven or eight weeks. During the past three months the intervals had grown shorter. Upon X-ray examination in the Out-Patient Department of this hospital the gastro-intestinal tract was negative. Plates of the gall-bladder region taken April 14 showed an indefinite shadow which might be stone or foreign material in the colon. At another examination April 21 the shadows were pronounced undoubtedly gall-stones.

Upon examination she was obese. The heart showed a soft systolic murmur at the apex. Pelvic examination showed the fundus pulled up to the anterior abdominal wall. Rectal examination showed two external tabs. No mass protruded on straining down. The reactions of the left pupil were more sluggish than those of the right.

Before operation the chart was normal, the urine and blood were not recorded.

April 30 operation was done. She was comfortable after it and by May 2 had no fever. There was a slight burn on the right buttock. There were still two or three protuberant external tabs at her discharge May 8.

After leaving the hospital she had no more bleeding until July 3, when she had a little bright red blood with a movement.

July 7 she reëntered for treatment of pain in the region of the right scapula of five years' duration and gastric discomfort of six months' duration. She now said that the pain had rarely come on while she was in bed at night. It started as a burning feeling, then became dull with sharp twinges. Heat or pressure almost always relieved it. It did not radiate. The attacks had increased in frequency and severity until she now had one almost every day. The epigastric pain was dull, non-radiating, occurred always about half an hour after meals, lasted about an hour and was relieved by doubling up. She had always had an attack if she departed from a bread and milk diet.

Upon examination she was still obese, though she had dieted and lost considerable weight recently. No heart murmurs were made out. The liver edge could be felt just below the costal margin. The left knee-jerk was greater than the right.

Before operation the temperature was 98° to 99.9°, the pulse 120 to 71. The respirations were normal. The urine showed a few red blood corpuscles and leucocytes, specific gravity 1.020. The blood is not recorded.

July 10 operation was done. The patient made a satisfactory ether recovery and next day was comfortable and looked well. The dressing stained through once the first day. The night of July 12 there was profuse bleeding. The pulse rose from 80 to 130 and the patient was restless, thirsty and sweating. It was found that no more blood came from the wound, so morphia was given and fresh gauze

applied. The pulse gradually returned to normal. About midnight the following night there was again bleeding through the dressing. The pulse rose to 120. At two a. m. it was 132 and she was in bad condition. Transfusion was done and the wound was opened and packed with gauze. One clamp was removed. In the morning the pulse was 120 and she was in fair condition. A duodenal tube through the nose was draining old bile.

July 13 a second operation was done. July 14 she died.

Discussion

By Dr. Edward L. Young, Jr.

If we can take the description of her bleeding as true she did have a very considerable amount of loss of blood. It is astonishing the amount of punishment that some people will stand before they go for help. Of course we always have to take statements of this kind with a grain of salt, because they may be very much exaggerated.

She has apparently two distinct problems. In the first place she had bleeding from the rectum said to be due to hemorrhoids, which had been present for fifteen or twenty years. Of course hemorrhoids can last for that length of time, but no one should make a diagnosis of hemorrhoids without examination. It is altogether too commonly done. And at twenty-five she presumably would not have carcinoma of the rectum.

The fact that she has had colitis makes us wonder whether or not there could be any trouble left in the colon to account for bleeding, or whether the hemorrhoids really are the whole story. There is no reason to assume that hemorrhoids degenerate into carcinoma. Of course carcinoma comes in the same region and can come after hemorrhoids have been present for a great many years. Whether the chronic irritation can be said to be a causative factor or not I think is doubtful, because it is very seldom that the combination occurs. Nevertheless the amount of blood which can be lost from hemorrhoids is of itself enough to justify interference even when there is very little discomfort from their presence, and apparently this woman did have constant loss of blood with at times a marked amount of anemia.

The X-ray shows gall-stones. It is a question of what treatment she deserves first.

The condition of the fundus was due apparently to a suspension which she had at the time she had her appendix out.

Is there any note of rectal examination other than this statement? They did not proctoscope to see if there was any ulceration above?

Miss Painter: There is no record of it.

Dr. Young: I do not see that we have anything to say other than that they considered the cure of the hemorrhoids the first thing.

Dr. Young's Pre-operative Diagnosis

Hemorrhoids.
Cholelithiasis.

Pre-operative Diagnosis, First Admission

Internal hemorrhoids.

Operation

Gas-ether. Under ether the sphincter was dilated to admit three fingers. This was accomplished only with great difficulty and by patient work. Two small hemorrhoidal masses were clamped and cauterized. An external tab was cut away and the skin edges united.

Further Discussion

Dr. Young: It does not sound as though there was a lot of trouble here, and if they had this same history in front of them that we have here I think they might have wondered whether there was anything going on above.

Of course it is too long to say that the red blood was due to the healing of the wound. It is a question whether it was another diffuse bleeding from the hemorrhoidal veins or whether it came from higher up. In view of the story that we have of trouble for so long and colitis, we wonder whether it comes from ulcerations in the colon. It is true that ulcerations in the colon can be present without more symptoms than this. A small amount of bright bleeding I think it is fair to say cannot come from high in the intestinal tract. Any massive bleeding which simply pours down through the intestines can appear, without being digested and without showing change, as bright blood, but it is unusual and I think here we must assume that it comes from lower down. However, there is only one note of it and it may be of no significance at all.

We have some laboratory work that helps us in this differential, that is the X-ray report that

the gastro-intestinal tract is negative and that there are undoubtedly gall-stones. Of course it is true that sometimes the history does not accurately differentiate between gall-bladder disease and peptic ulcer. I think it is true that a great many histories where the difference is not clearly brought out are histories written without sufficient care. In a great many cases careful questioning of the patient will bring out the story pretty accurately, and of course it has been said that diagnosis of duodenal ulcer can be made on the story alone in ninety-nine per cent. of the cases. But I think it is one of the differential diagnoses where an accurate history is of as much importance as it is in almost any other condition.

DR. MAURICE FREMONT-SMITH: I have seen a couple of cases of attacks of intense pain which were apparently gall-bladder pain, having no relation to meals, coming out of a clear sky, not coming every day, in which a duodenal ulcer with massive adhesions was found. You have probably seen the same.

DR. YOUNG: I think the two things can exist together.

There is just one other thing to emphasize, and that is that an X-ray report should never be accepted as authoritative unless it does agree with the clinical findings. That is, if one is certain that the history is ulcer, and the X-ray says no ulcer, the X-ray should not be accepted as final on one examination.

I assume from what we know that at operation the gall-bladder with stones was removed.

DR. YOUNG'S PRE-OPERATIVE DIAGNOSIS

Cholelithiasis.

PRE-OPERATIVE DIAGNOSIS JULY 10, SECOND ADMISSION

Cholelithiasis.

OPERATION

Gas-ether. Transverse gall-bladder incision. The gall-bladder contained several stones. It was removed from below up. The cystic artery and duct were clamped and tied. There was considerable bleeding from the liver which seemed to come from a large vein. This was clamped in several places. The clamps were left on and two gauze sponges packed against them.

PATHOLOGICAL REPORT

A dilated gall-bladder filled with thick brown fluid with two greenish facetted stones the size of cherry pits. The mucous membrane is smooth.

Chronic cholecystitis.
Cholelithiasis.

FURTHER DISCUSSION

It is unusual to have this amount of bleeding coming from the bed of a gall-bladder which was taken off in a routine way. It is not usual that clamps are left on in that fashion.

Here is a death from hemorrhage, but not due to the oozing of a jaundiced patient. It is not usual that this cause of hemorrhage is present in this operation. We often have to consider the question of oozing due to the low coagulability of the blood in jaundiced patients, but very seldom this condition.

In all hemorrhage we have to consider not only that the patient may die of hemorrhage but that even if the hemorrhage is not severe enough to kill the lowering of local and general resistance from moderate bleeding may open the way to infection which itself kills. I think that is a fact which is often lost sight of in surgery. It was first emphasized to me in prostatic surgery where it was easy to say, "The patient is not bleeding much," and yet the patient died, of low-grade sepsis, of pneumonia, and I believe a great many of those cases would not have become septic if they had not lost enough blood to pave the way for the entrance of infection. I think in a great many bleeding cases in the abdomen the same thing may be true. Here I think Dr. Richardson will tell us that there is massive hemorrhage into the abdominal cavity.

We have nothing to go on to assume that there was anything in the colon. Of course bleeding for twenty years cannot come from carcinoma. But the diagnosis of colitis so-called is so uncertain, the symptoms are so varied and I think Dr. Richardson will agree that he often finds it present where there have been no symptoms—it seems possible that he may report damage in the colon as a cause of that bleeding. Of course a chronic colitis which had been present would by this time have shown chronic inflammatory reaction with pus and blood. I do not think we have any right to say carcinoma, but I think there will be some pathology other than the old hemorrhoids.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Cholelithiasis.
General peritonitis.
Operations, laparotomy, cholecystectomy with drainage for secondary hemorrhage.

DR. EDWARD L. YOUNG'S DIAGNOSIS

Cholelithiasis.
Hemorrhage.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

(Chronic cholecystitis.)
(Cholelithiasis.)

2. *Secondary or terminal lesions*

Slight icterus.
Thrombosis of the inferior vena cava and left iliac veins.
Distension of stomach and intestines.
Dilatation of the heart.
Soft spleen.
Anemia.

3. *Historical landmarks*

Scar of old operation wound.
Slight chronic pleuritis.
Lipoma of the small intestine.

DR. RICHARDSON: There was slight icterus.

The operation wound in the anterior abdominal wall extended across it and was closed with sutures except at the right end, where it was open and wicks protruded. The abdomen was moderately distended, the wall slightly tense. There was a good amount of subcutaneous fat.

The peritoneal cavity contained a small amount of fluid blood and blood clot, apparently what was left after the evacuation of blood. The peritoneum along the coils of the small intestine here and there was blood-stained. The stomach was considerably distended, but the mucosa and pylorus were negative. The intestines on section were negative. There was some injection of the vessels of the wall. At a point about 150 cm. above the ileocecal valve a small fibrous-like cord hung off the wall of the small intestine. At the end of that there was a small spherical mass looking something like fat. There was no lumen in the cord, and at the point where it was attached to the wall of the intestine there was no dimple on the mucosal side. Of course the investigation was to rule out a Meckel's diverticulum. It was nothing but a small lipoma.

The margin of the liver was three cm. above the costal border. The diaphragm was at the third interspace on the right, at the fourth on the left.

There were a few slight pleural adhesions on each side. There was a little edema of the lung tissue.

The heart weighed 260 grams, with a flabby pale myocardium, negative valves and free coronaries. Extending from the first portion of the inferior cava down along the left iliac veins there was a frank thrombus, a source of course for pulmonary embolism, which she did not have. Below the lesser omentum the radicles in the mesentery were injected. The packing for the hemorrhage pressed rather markedly on the duodenum, the pyloric end of the stomach and the lesser omentum. It probably pressed somewhat on the portal vein and caused the injection of the radicles below. There was also much distension of the stomach above this point of pressure. At one place on the liver a pair of hemostatic forceps was snapped to stop hemorrhage. The liver was otherwise negative.

DR. YOUNG: Could you tell whether there was a vein?

DR. RICHARDSON: Where the snaps were applied the liver surface was eroded. The gallbladder was wanting. The cystic duct was tied off, negative, and the hepatic and common ducts negative.

The spleen was rather soft. There were three small myomata in the wall of the fundus of the uterus. Extending from the top of the uterus was a broad band of fibrous tissue which fastened the uterus to the anterior parietal perineum,—I presume a ventral fixation. The appendix was negative. Apparently they did not take it out.

There was no growth in the blood culture from the heart. Microscopic examination confirms what we have said.

A PHYSICIAN: What could have been done to stop the hemorrhage?

DR. YOUNG: Hindsight is always easier than foresight, of course. I think that a large liver needle carrying a suture well around the bleeding area and tied over a bit of muscle, or even fat, might have stopped it. I do not know that one could have done that here, because this is an unusual form of accident in cholecystectomy.

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STOMATOLOGY OR DENTISTRY?

THE relation of dentistry to general medicine is one of the problems under discussion today. Most of us feel that the dentist has certain well-defined duties in regard to the welfare of the individual, and he is carrying out these duties both to his profit and to that of his patients. It appears, however, that in the opinion of a number of men well qualified to hold views on this subject, the present situation is deplorable. Dentists, they maintain, are merely mechanics, whereas they should be doctors. In other words, dentistry is a branch of medicine, and those who practise it should be rated as medical specialists. The specialty of stomatology, which shall embrace diseases of the mouth and teeth, both medical and surgical, should be developed. It should include odontology, or the care of teeth, and all dentists should become stomatologists.

In order to bring about the realization of this ideal, the American Stomatological Association advocates the following changes:

1. To establish stomatology as a specialty of medicine, to be practised by graduates in medicine and stomatology.

2. Until the law requires the M.D. degree for the practice of dentistry, and during this transitory period, arrangements will be made as soon as possible, for licensed practising dentists, holding the D.D.S. or D.M.D. degrees from reputable institutions to be granted the M.D. degree, after suitable preparation and qualification.

3. To gather clinical and experimental data pertaining to the scientific problems of stomatology.

4. To encourage research in stomatological problems, by providing the necessary funds.

5. To see that all members of the American Stomatological Association automatically become members of the American Medical Association. To arrange sections in stomatology in all local medical societies.

6. To work out plans for carrying on instruction in clinical stomatology by physicians and dentists, in existing medical and dental institutions, and to arrange a curriculum suitable for present and future needs.

7. To reorganize, from the stomatological point of view, the available information pertaining to mouth hygiene.

These proposed changes are in accord with the resolutions passed by the French Association of Dental Surgeons, and with the situation which already exists, so far as we can ascertain, in Italy.

So radical a departure as this, sponsored as it is by men of standing in their profession, deserves careful consideration. We can readily believe that there is need for a limited number of doctors of medicine who have specialized in diseases of the mouth, to whom doubtful cases may be sent for consultation. Such men may give lectures on the broader aspects of odontology to dental and medical students, and by their continued interest, encourage research in problems relating to the teeth and jaws. We are rather appalled, however, by the thought of imposing upon thousands of good dentists the necessity for undergoing further studies in order to gain a degree of M.D. which will not permit them to practice medicine. Many a prospective dental student, it would seem, would hesitate before investing the additional time and money required to give him a medical degree and one year in a hospital, plus his technical training in odontology. We believe, though admittedly without definite knowledge of the subject, that at least nine-tenths of the work required of dentists is of such a nature that it can be performed with perfect satisfaction with the education already provided in good dental colleges. In such schools, the fundamental sciences—anatomy, physiology, bacteriology and pathology—are taken up in detail, and the relation of odontology to the organism as a whole is certainly made clear. It may be mentioned in passing that the Section of Stomatology of the American Medical As-

sociation has been given up because of poor attendance at the Annual Session.

We fear that the result of the changes in the status of the dentist as desired by the American Stomatological Association would in truth be deleterious to the public health. Fewer men would enter dentistry, because of the increase in cost and because of the attraction to those other fields of medicine which would be open to them. The cost of dental services, already burdensome, even though warranted, would be increased, thereby driving more patients to the free clinics, or leading them to neglect their teeth.

We suspect that the feeling of social inferiority which is believed by some to attach to the profession of dentistry as compared with that of medicine has considerable to do with the desire to place the dentist on a plane with the specialist who has an M.D. degree. Whether or not such a distinction really exists we cannot say. There are socially charming dentists and socially impossible doctors. It seems probable that in the long run, every one secures that standing, social as well as professional, to which his parts entitle him. We wish no injustice to the doctor of dental surgery, who has indeed our heartiest respect and often our profoundest gratitude. Would he be more able to help either humanity or himself if the letters after his name were changed?

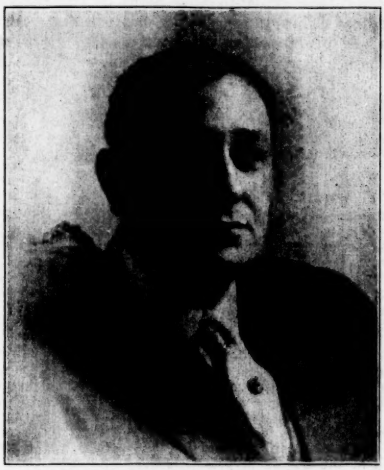
THE MEETING OF THE FOUR WESTERN DISTRICT MEDICAL SOCIETIES

THE annual group meeting of the Berkshire, Franklin, Hampden and Hampshire District Medical Societies was held at the Hotel Kimball in Springfield, November 12, 1925.

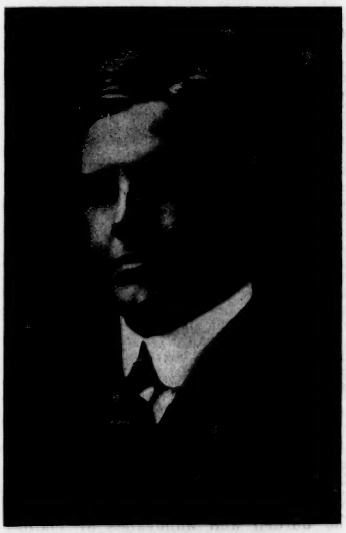
An excellent luncheon was served at one o'clock, and after this was finished the President of this group organization, Dr. A. P. Merrill, called the meeting to order, suggested the continuation of the organization, and called for the election of a president for the ensuing year.

Dr. George Dallas Henderson of Holyoke was elected President and Dr. Hervey L. Smith, Secretary.

Dr. James S. Stone, President of the parent society, was the first speaker. He outlined the activities of the Massachusetts Medical Society especially emphasizing some of the features which are designed to be of service to the members. He was especially pleased with the plan to hold the next annual meeting in Springfield for it is confidently expected that the success of the meeting in Pittsfield will be duplicated. The meeting will be held in the Hotel Kimball which is well equipped to accommodate the Society. There will be a resumption of the custom of several years ago in having scientific and commercial exhibits. Assurances have been received that exhibits covering the work in Diabetes, Tropical Diseases and Fractures will be held and there will probably be exhibits setting forth the problems of Heart Disease, Cancer and Nutrition. The annual dinner will be at one-thirty on Wednesday, and after the Shattuck Lecture Tuesday evening, Dr. George



DR. GEORGE DALLAS HENDERSON



DR. HERVEY L. SMITH

B. Magrath will give an interesting address based on his experiences as Medical Examiner of Suffolk County.

Dr. Stone explained the plans of the Boston Medical Library to provide medical literature for the profession as set forth in the following letter:

BOSTON MEDICAL LIBRARY

November 11, 1925.

Dr. J. S. Stone, President,
Massachusetts Medical Society,
Boston, Mass.

Dear Sir:

Concerning your request for information regarding the service which the Boston Medical Library renders to its members residing outside of Greater Boston.

The fee for associate membership is six dollars a year and for this fee the member is entitled to borrow three books or periodicals, which may be retained for one week. All transportation charges are paid by the member borrowing the books.

Personally I have been looking forward to the time when it would be possible to establish branch libraries in the cities remote from Boston. Any scheme which would make this plan successful would have to have the coöperation of the physicians of the particular locality where the branch was established, of the institution where the library would be located and of the Boston Medical Library.

I have not worked out a definite plan of organization and operation, but my thought has been along the following lines: First, the physicians of the locality would have to get together and support the plan as a unit. They would become associates of the Boston Medical Library and their fees would be used to supply books and periodicals for the branch. The district society would be asked to contribute towards the operating expenses, which would include transportation expenses.

The institution housing the collection, preferably a library, would give stack and reading space and supervision.

The Boston Medical Library would select the books, do the cataloging and build up a select reference library of books and standard sets, utilizing its duplicates for this purpose. The Boston Medical Library would lend as occasion demanded the material required for special research. The Boston Medical Library would supply at cost lists of references on particular subjects as desired by members.

The title in the books would rest with the Boston Medical Library and only members would be permitted to borrow for home use.

Such a plan is possible, but only with the full and hearty coöperation of all concerned.

It is to be understood that this is my personal opinion and not binding on the Boston Medical Library, as the authorities of the Boston Medical Library have not acted upon the matter.

Yours very truly,
JAMES F. BALLARD,
Assistant Librarian.

Brief reference was made to the plans of the Society relating to Health Examinations.

The burden and annoyance of malpractice suits, most of which are blackmail, was referred to. We must, however, recognize that there may at times be some reason for these claims. He informed the meeting that a committee is now working on the questions relating to indemnity insurance and expected that steps will be take to protect the members against unjust suits. He felt that the adoption of the recommendation of the Society to make use of the group insurance plan made operative by Mr. Crosbie has provided a service of value and should be endorsed pending a report of the committee. Future position of the Society may depend on the report of this committee. Warning the members that many suits result from some action or statement made by a doctor, he urged that great care should be exercised to avoid saying or doing anything which might stimulate a dissatisfied person to start a suit.

Referring to the action of the Massachusetts Medical Society in depriving homeopathic practitioners of membership in the Society many years ago, Dr. Stone was very glad to announce that the only surviving member of this group had been reinstated and that the future will show still greater cordiality and coöperation by the Massachusetts Homeopathic and the Massachusetts Medical Societies. Both societies have worked harmoniously in legislative matters for many years and this last element of dissatisfaction has been eliminated.

Hope was expressed that the work of the legislative committees will be augmented by the Boston Chamber of Commerce which has indicated a definite interest in all activities tending to promote health. We must, however, do all in our power to assist any effort which may be made by lay organizations.

Taking up the matter of the registration of physicians, attention was called to a letter sent by the Legislative Committee to all members of the Society explaining the wisdom of removing the restrictions now imposed upon the Governor in making appointments to the Board of Registration in Medicine, and the importance of maintaining a single standard for all practitioners of medicine.

He explained the essential principle of the present law and urged all members of the Society to make the strongest possible contest against any deviation from the position taken by the state, for multiple examining boards mean multiple standards which would inevitably creates in this state the disgraceful situation recently existing in Connecticut, but which has been corrected to a large extent by the demand of the people. We must keep clearly in mind that it should not be the function of the state to dictate as to treatment but should demand that all practitioners must have an understanding of the basic sciences on which the structure of medical practice is built.

He stressed the importance of supervision of medical schools to the end that students may be assured of adequate equipment in those institutions devoted to the teaching of medicine. Allusion was made to the fact that of the five substandard schools two are in Boston and in

addition, the Massachusetts College of Osteopathy is also located here. Graduates of these schools are accepted for examination only in the District of Columbia, Nevada and Massachusetts. All other states do not recognize these institutions as qualified to teach medicine. The records of the Massachusetts Board of Registration in Medicine show that in the period 1911 to 1924, of graduates of the Harvard Medical School only 1.14% were rejected, of Boston University Medical School 4.83% were rejected and of Tufts College Medical School 7.5% were rejected, while during this period, of 303 graduates of the Massachusetts College of Osteopathy 57.4 were rejected, of 156 graduates of the Boston College of Physicians and Surgeons 65.7% were rejected and of 160 graduates of the Middlesex College of Medicine and Surgery 49.3% were rejected. Dr. Stone raised the question as to the justice of allowing schools to continue in operation with records such as those shown in the list of these mentioned. These problems should be the individual concern of every public spirited citizen. In this connection he quoted Dr. Pusey, formerly President of the A. M. A., who feels that the general scheme of medical education should be so modified that while only well qualified men should be allowed to practice, more attention should be given to the training of general practitioners rather than to scientific investigation so that the people at large may have a larger available supply of doctors.

The Society should feel responsible for any deviation from the original purpose of medical colleges which is to provide the greatest possible number of practicing physicians, for it may be that the cost of medical education is made greater by reason of the expense of unproductive research and is tending to discourage those persons who need to develop earning capacity earlier than is the case with many who enter upon the practice of medicine under present conditions.

Dr. Haven Emerson then addressed the meeting, taking as his subject "Health Examinations." Having been occupied for a considerable time in dealing with public health matters he had come to feel that public health is a private responsibility. After giving a brief history of some experiences which led up to modern public health activities previous to 1900 we then entered upon another phase characterized by more general participation by larger numbers and groups of intelligent people. The first individual engaged in public health work was appointed by the Appellate Court of Connecticut.

The newer method was especially exemplified by the interest in the problems of tuberculosis, leading up to more definite measures ralating to the control of communicable diseases.

Reference was made to other features of preventive medicine using the experience of the New York Board of Health in dealing with infant mortality, heart disease and social hygiene.

Another stage sponsored by the A. M. A. and National Health Council was entered upon in 1922 when the health propaganda took on the scheme of studying the health of individuals by competent diagnosticians.

While the history of the first attempts to deal with tuberculosis showed that the study encountered only the advanced cases which were doomed to die, later in one of the large clinics to which people were invited, thirty thousand persons were found to be free from evidence of tubreculosis. The great advance in promoting health of the individual will come, not through existing organizations, but by the relation of the individual to the private practitioner by means of periodic health examinations.

This method is now generally accepted as the most important threshold of the third era.

The theory is to get ahead of the fixed disease. It has been shown that definite reduction in mortality results from the study of the expectant mother, children and the person in industry. The important demonstration will be in ascertaining whether the examination is more valuable than the opinion of the patient. It must be recognized that an examiner who is unable to diagnose a disease is not competent to conduct a health examination, for the procedure is no different from that taught in the medical schools or required in any general hospital. The object is to find some defect or to assure the patient that none exists.

This work as applied to the intelligent study of the pregnant woman shows a reduction of fifty per cent. of deaths in the first and second months of life and extending this study to the child up to two years of age teaches how to bring up a healthy baby. The medical care of babies already sick does not accomplish much in saving life but the well baby must be kept well.

By this means the mortality of infancy has been cut in half and again still further cut in half. This does not mean new knowledge but simply means delivering the goods.

During the pre-school age the average parent is stupid and if not advised the child will be neglected and will enter school with obvious defects. The important thing is to deliver the child to the school free from defects for the school health service is superficial and only takes cognizance of obvious troubles. Regular examination of the pre-school child eliminates many handicaps and fewer children are held back.

The periodic examination of employees is an economic as well as a health measure yielding appreciable dividends, for unrecognized cases of diabetes, heart disease, renal and mental dedefects are found. Health examination should

be made every six months in the pre-school age, once a year during school life. In women the tuberculosis hazard is especially prominent between the ages of fifteen and twenty-five and this class should be carefully guarded by frequent examinations. All adults should have a yearly examination up to sixty-five and oftener if any lesion exists and after sixty-five every six months.

The health examination should take thirty minutes or more and the fee should be at least five dollars. The doctor must learn how to make an examination, for his experience has been so constantly with disease that he is seldom qualified to differentiate health and slight or early deviations. The big job with many physicians will be the acquisition of the technic of a thorough examination.

Dr. W. P. Bowers presented samples of slips which will appear in the BOSTON MEDICAL AND SURGICAL JOURNAL and may be used to record the results of the examinations. It was intended to avoid the long complicated forms which have been recommended by some organizations, for these blanks will be at least suggestive of details which each examiner will develop as he becomes more familiar with the work. Dr. Emerson felt that the blanks recommended by the A. M. A. would be found to be more valuable.

The meeting was successful and was attended by about sixty representatives of the Societies.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

BOWLER, JOHN POLLARD, A.B., M.D., Dartmouth 1915, M.D., Harvard Medical School 1919; M.S., University of Minnesota 1924; Instructor in Anatomy, Dartmouth Medical School; Member of the New Hampshire Surgical Club. The subject of his paper is "The Management of Obstructive Jaundice as a Factor Affecting Surgical Risk."

MCCLURE, CHARLES W., M.D., Ohio State University College of Medicine 1910; Chief of the Gastroenterological Research Division of the Department of Biochemistry and Gastroenterology to the Evans Memorial; Gastroenterologist to the Out-Patient Department, Homeopathic Hospital, Boston. Dr. McClure writes a series of papers on "Studies in Liver Function. 3. Methods for determining the furfurol number and the bilirubin concentration of duodenal contents. 4. A Procedure for the Uniform Stimulation of Biliary Flow. 5. Clinical Observations on the Evaluation and Treatment of Disturbed Liver Function."

Associated with Dr. McClure in the writing of these papers are:—

GOTTLIEB, JULIUS, A.B., Harvard; M.D., Boston University 1924; Resident Pathologist, Boston Homeopathic Hospital; Member, Mass. Surgical and Gynecological Society.

HUNTSINGER, M. E., Technician.

MENDENHALL, W. L., M.D., Drake Univ., College of Medicine, Des Moines, Iowa, 1906; Professor of Pharmacy, Boston University School of Medicine

and

MONTAGUE, O. C., Chemist and Physicist.

SHATTUCK, FREDERICK C., A.M., M.D., Harvard Medical School 1873; Emeritus Jackson Professor of Clinical Medicine, Harvard Medical School. His subject is "The Dramatic Story of the New Harvard Medical School."

BURNETT, FRANCIS L., S.B., M.D., Harvard 1906; Graduate Assistant in the Skin Department, Massachusetts General Hospital; Research in Nutrition, Massachusetts Charitable Eye and Ear Infirmary; Instructor of Nutrition, Forsyth Dental Infirmary. His subject is "The Progress of Nutrition."

The Massachusetts Medical Society

COLLEGES OF GRADUATION OF CANDIDATES FOR FELLOWSHIP IN MASSACHUSETTS MEDICAL SOCIETY, EXAMINATIONS OF NOVEMBER 5, 1925

Mr. Editor:

The Fellows will be interested in the following figures as to the colleges of graduation in medicine of the 97 candidates for fellowship that were passed on favorably by the Censors at the examinations held in the various districts of the state on November 5, 1925. It may be noted that under the terms of the By-Laws these do not become Fellows until their diplomas have been signed by the president and secretary.

Tufts College, 41.
Harvard University, 21.
Boston University, 5.
College of Physicians and Surgeons, Boston (action of Committee on Medical Education and Medical Diplomas), 4.
Jefferson Medical College, 5.
University of Pennsylvania, 3.
University of Maryland, 2.
McGill Faculty of Medicine, 2.
One each from the following:
University of Colorado; University of Vermont; Temple University (action of Committee on M.E. and M.D.); Georgetown University; State University of Iowa; University of Toronto; George Washington University; University of Maryland; Stanford University; Johns Hopkins University; Baltimore Medical College (action Committee on M.E. and M.D.);

St. Louis University; Medical School of Maine (action of Committee on M.E. and M.D.); Cornell University.

WALTER L. BURRAGE,
Secretary.

MISCELLANY

THE CHRISTMAS SEAL SALE

CHRISTMAS Seal slogans over the air will be a novel feature of the eighteenth annual Seal Sale which began Friday, November 27. Dr. John B. Hawes, President of the Boston Tuberculosis Association, will broadcast from the Shepard Stores a brief statement of the work of the Boston Tuberculosis Association and urge the people to buy Christmas Seals. Frederic Edwards, Executive Secretary of the Hampden County TB Association, has arranged through the courtesy of WBZ of Springfield to broadcast nightly pithy statements as to the part the Sale of Christmas Seals has played in reducing the deaths from tuberculosis in this country by more than half. During the week Mr. Edwards will broadcast the story of the Christmas Seal. Eighteen years ago a Danish postal clerk conceived the idea of raising funds to help sick children through the sale of Seals at Christmas time. It was an instantaneous success in Denmark and the idea was shortly carried over to this country by Miss Emily Bissel of Delaware. The struggle of the early years of Miss Bissel's efforts and her success in getting the seal widely adopted so that now over five hundred million seals are sold annually at Christmas time for anti-tuberculosis work is an epic in American philanthropy.

Frank Kiernan, Executive Secretary of the Massachusetts Tuberculosis League, announced today that all the Christmas Seal Campaign material has been dispatched from the League Headquarters in the Little Building, Boston, to the twenty-seven affiliated organizations throughout the State. Mr. Kiernan has given interesting talks to large groups of volunteer workers in all counties of Massachusetts. Last week Northampton, Lawrence, Worcester, Taunton, Framingham, Beverly, Boston and Brookline were included. There has been in the past week an insistent demand for Seals in all places visited. The League has had to send to New York, Minneapolis, and Scranton for additional supplies. The cry for Seals has never been so great in the history of this Massachusetts organization.

Tuberculosis workers all over the State credit the demand this year for increased amounts of Christmas Seals to the fact that in every section of the State last summer camps were provided to care for children exposed to the disease. In all over two thousand children were cared for and the aggregate gain in weight was two thousand pounds.

The Boston Tuberculosis Association at 25 Huntington Avenue is the local representative of the Massachusetts Tuberculosis League.

THE SEAL SALE IN BOSTON

MRS. A. KATHERINE MCGUINNESS, who is in charge of the booth sale of Christmas Seals for the Boston Association of the Massachusetts Tuberculosis League, has submitted a temporary report of those in charge of the various booths throughout the city. Those in charge of the booths are as follows: Mattapan Post Office—Miss Madeline Homold; Huntington Ave. P. O.—Mrs. Julius Levine; Fields Corner P. O.—Mrs. D. Frank Doherty; Charlestown P. O. and Boys' Club of Boston—Mrs. B. A. Whittle; Army and Navy Y. M. C. A.—Miss Elizabeth Brown; Upham's Corner P. O.—Mrs. Lloyd Kelly; Fenway P. O.—Miss Emily Boswell; Hyde Park P. O.—Mrs. Charles F. Stack; West Roxbury P. O.—Mrs. Gerardi Balboni; East Boston P. O.—Mrs. Rose Valletta; Roxbury P. O.—Mrs. Sweetser; Blackington Mothers' Club—Miss E. L. Osgood; Jamaica Plain P. O.—Mrs. Goodrich; Brighton P. O.—Mrs. John Gallagher; Adams House—Mrs. W. W. Fogg; Hotel Lenox—Mrs. T. F. Dickinson; Touraine—Mrs. Wm. Hirshon; Dutton's Roxbury Store—Mrs. John B. Hall; Symphony Hall—Mrs. Nash; Dorchester Woman's Club, Codman Sq. Booth—Mrs. Minor H. A. Evans; Marston's—Miss Anna Maltey.

Many other booths to be opened will be announced later. All booths, with the exception of those in the various hotels will open on December 2. The hotel booths will open about a week before Christmas. The Boston Tuberculosis Association expects the biggest booth sale of Christmas Seals ever held in its history.

Many of the most prominent women of Boston are enrolled in its cause.

NEWS ITEM

TUFTS MEDICAL SCHOOL—A "smoker" was given by the junior class of Tufts Medical School, November 5. About 70 were present, including 14 members of the faculty, several of whom gave short talks. An enjoyable evening was spent by all.

REPORTS AND NOTICES OF MEETINGS

CLINICAL EXERCISES AT THE MASSACHUSETTS GENERAL HOSPITAL

ON Thursday, December tenth, at 8:15 p. m. there will be the third monthly meeting at the Massachusetts General Hospital. The program will consist of:

1. Demonstration of cases.

2. Dr. Chester Jones will speak on "Chronic Biliary Disease Following Apparently Benign Jaundice."

3. Dr. Richard Cabot will speak on Cardio-Vascular Disease, Clinical and Post-mortem records for twenty years.

Doctors, medical students and nurses are invited.

PHYSIOLOGICAL CONFERENCE

THE sixth weekly meeting of the Physiological Conferences will be held in the Bowditch Library of the Harvard Medical School on Monday afternoon, December 7, at 4 o'clock. Dr. J. F. Fulton will speak on "Inhibition of the Knee Jerk and the Nature of the Inhibitory Process."

NEW ENGLAND PEDIATRIC SOCIETY

THE ninety-third meeting of the New England Pediatric Society will be held at the Boston Medical Library on Friday, December 11, 1925, at 8:15 P. M.

I. The report of the Treasurer.
II. The report of the Council.
III. The following papers will be read:
1. The Mechanism of the Action of Sunlight on Development
William T. Bovie, Ph.D., Boston
2. Some Uses of Ultraviolet Light in Diseases of Children
Edwin T. Wyman, M.D., Boston
IV. Election of Officers.

Light refreshments will be served after the meeting.

KENNETH D. BLACKFAN, M. D., *President.*
JOSEPH GARLAND, M.D., *Secretary.*

THE LAWRENCE MEDICAL CLUB

THE monthly meeting of the Club was held on Monday evening, November 24, with Gustave E. Kurth, M.D.

Chairman for the evening, Dr. Gustave E. Kurth, M.D. Subject: "Diagnosis and Treatment of Some Orthopedic Conditions," Benj. E. Wood, M.D., of Boston.

Organized at eight-thirty o'clock.

The legislative plans of the Massachusetts Medical Society were outlined and the coöperation of the members of the Club was secured.

R. V. B., *Secretary.*

CLINICAL AND SURGICAL ASSOCIATION OF MASSACHUSETTS

THE Fall Convention of the Clinical and Surgical Association of Massachusetts was held at the Johns Hopkins Hospital, Baltimore, Md., on November 3rd and 4th. Twenty members were present and were splendidly entertained by the staff of the Johns Hopkins Hospital.

THE NORFOLK DISTRICT MEDICAL SOCIETY

A REGULAR meeting of the Society was held in Roxbury Masonic Temple, 171 Warren Street, Roxbury, November 24, 1925.

Business:

Communication: "Some Aspects of Thyroid and Adrenal Failure," Dr. Charles H. Lawrence.

Discussion opened by Drs. J. H. Means and Joseph C. Aub.

Collation.

FRANK S. CRUICKSHANK, M.D., *Sec.*

AMERICAN BOARD OF OTOLARYNGOLOGY

AN examination was held by the American Board of Otolaryngology on October 19, 1925, at the Cook County Hospital, Chicago, with the following result:

Passed	120
Failed	23
Total Examined	143

The next examination will be held in Dallas, Texas, on April 19, 1926. Applications may be secured from the Secretary, Dr. H. W. Loeb, 1402 South Grand Boulevard, St. Louis, Missouri.

SOCIETY MEETINGS

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

January 6, 1926—The semi-annual meeting at Haverhill.
May 5, 1926—The annual meeting at Lawrence.

Essex South District Medical Society

Wednesday, January 6—Beverly Hospital. Clinic, 5 P. M. Dinner, 7 P. M. Speaker, Dr. Paul D. White, Boston. "Recent Progress in the Study and Treatment of Heart Disease."

Wednesday, February 3—At 7 P. M. Hawthorne Hotel, Salem. Dr. Walter Timme, New York. Subject to be announced.

Wednesday, March 3—Lynn Hospital. Clinic, 5 P. M. Dinner, 7 P. M. Dr. Charles E. Mongan, Somerville, "Some Problems of Present-Day Practice."

Thursday, May 6—Censors meet at Salem Hospital, 3:30 P. M.

Tuesday, May 11—The Tavern, Gloucester. Annual meeting. Speaker to be announced.

Middlesex East District Society

January 13—At the Harvard Club at 6:30 P. M. Address by Dr. Richard Ohler, "Metabolism."

February 10—At the Harvard Club. Address by Dr. William F. Boos; subject, "Industrial Poisoning."

April 14—At the Harvard Club at 6:30 P. M. Address by Dr. William E. Ladd, subject to be announced later.

May—Annual meeting, Colonial Inn, North Reading. Subject and speaker to be announced.

Suffolk District Medical Society

January 6—At 8:15 P. M. Medical Section (meeting postponed from December). Dr. W. J. MacDonald will speak on "Experimental Work in High Blood Pressure."

January 27—At 8:15 P. M. Combined meeting with Boston Medical Library. "Medical Experience of an Explorer," Dr. J. Hamilton Rice

February 24—At 8:15 P. M. Surgical Section. "Post-operative Care of Surgical Cases," Dr. Dean Lewis, Chicago. "Surgical Convalescence," by Dr. John Bryant.

March 31—At 8:15 P. M. Medical Section. Subject to be announced later.

April 28—At 8:15 P. M. Annual meeting. Election of officers. "Some Diagnostic, Prognostic and Therapeutic Aspects of Disorders of the Blood," Drs. George R. Minot, Cyrus C. Sturgis and Raphael Isaacs.

Notices of meetings must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.